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TRANSACTIONS & STUDIES
of the
College of Physicians of Philadelphia

Volume 26
(Fourth Series)

Number 1
(June 1958)

The So-Called Hypersplenic Syndrome: Its Pathogenesis, Recognition and Management[1]

By R. PHILIP CUSTER, M.D.

THE honor and privilege of delivering this, the Seventieth Thomas Dent Mütter Lecture, is deeply appreciated. The will of Doctor Mütter set a precedent at the College of Physicians of Philadelphia in creating a named lectureship. At the same time, he bequeathed the nucleus of a museum of pathologic anatomy and endowed it substantially, a museum that has become even more renowned for its treasures of medical history and curiosity, especially in recent years under the able curatorship of Mrs. Ella Wade.

Doctor Mütter must have been an exceptional person. Born in 1811 and orphaned at the age of eight, he obtained his doctorate in medicine at the University of Pennsylvania twelve years later, studying under Dupuytren, Louis, and Liston during the following year. Details of these early years are not available, but one can be certain that they were not easy ones for him. He commenced the practice and teaching of surgery in Philadelphia in 1832, and was appointed a professor of surgery at the Jefferson Medical College in 1841, achieving distinction for pioneer work in plastic and reconstructive procedures. Ill health forced his retirement in 1856, and he died three years later.

During my term as a member of the Committees on the Mütter Museum and College Collections, I have often thought that Thomas Dent Mütter must have been greatly influenced by the elder Gross (1805–1884), his contemporary at Jefferson, at heart a pathologist although cloaked as a surgeon. Samuel D. Gross published the first American treatise on pathology ("Elements of Pathological Anatomy," Boston, 1839), and gave the first organized courses on the subject in the United States. He might well have stimulated Mütter to collect and mount in his home the specimens that led to the Museum which bears his name. In connection with the Lectureship, Doctor Mütter also specified that it be concerned with "some point or points connected with surgical pathology." A consideration of splenic disorders that frequently require removal of that organ should fulfill his stipulation.

In preparing the material for this presenta-

From the Laboratories of the Presbyterian Hospital in Philadelphia, and the Department of Pathology, University of Pennsylvania School of Medicine.

[1] Thomas Dent Mütter Lecture LXX, College of Physicians of Philadelphia, 11 December 1957.

tion, however, it became quite obvious that I had bitten off far more than I could chew or you could swallow in one evening. A revised plan will still encompass the general field of splenic dysfunction, but the better known aspects will be considered in a very brief fashion.

Somewhat more attention will be given our experience with so-called secondary hypersplenism in connection with malignant disease of the reticulo-endothelial system, i.e. the lymphomas, leukemias, and allied disorders. Emphasis is laid on the frequent failure of physicians to recognize or even consider the possible role of the spleen in causing the serious anemia, neutropenia, and/or thrombocytopenia that so frequently arise during the course of these diseases. Likewise, it would be well to counteract, if possible, the traditional timidity in having splenectomy performed because of the malignant background. The tendency has been to attribute these cytopenias to displacement of the bone marrow by malignant tissue, which seldom happens, or to destruction of the bone marrow by therapeutic agents, which happens all too often. Thus the physician is apt to stand by passively, when at least some of these patients could be salvaged.

For the painstaking tabulation of data on this series of cases, relatively little of which I will be able to show you this evening, I am greatly indebted to our Senior Fellow in Pathology at the Presbyterian Hospital, Doctor Bong Hak Hyun, and to Doctor James Butcher for his able partnership in the diagnosis and management of the patients. Our various Residents in Pathology over the years have likewise contributed immeasurably to the work, and I wish that time permitted expression of my appreciation of them by name.

THE MECHANISM OF BLOOD DESTRUCTION

Before considering the means by which the spleen plays a major role in bringing about significant cytopenias, it might be well to review for a moment the normal circulation of blood through that organ, which is still a controversial subject. While more evidence points to the vascular system within the spleen as being a closed one, there is no doubt as to the blood flow through it being exceptionally stagnant in comparison with other organs or tissues. It has been postulated, but not proven, that the spleen prepares erythrocytes for destruction during this period of stasis through the action of lysolecithin on the cell membrane. To me an unscientific but more attractive thought is an ability of the spleen to recognize in some fashion the red cells that are approaching senescence, and to dispose of them forthwith during their period of slow passage through the expanded sinusoidal bed.

Cells of the reticulo-endothelial system in the spleen and elsewhere are concerned with the products of blood cell breakdown, but under normal circumstances, and even under many conditions of hypersplenism, one does not see frank erythrophagocytosis, leukophagocytosis or ingested platelets, although hemosiderosis is frequently noted in acquired hemolytic anemia.

Abnormally functioning spleens in creating peripheral hematocytopenias can do so in one of several manners. The simplest is by *sequestration* of the missing elements of the blood. Perhaps the best example of this is found in hereditary spherocytosis, where erythrocytes at the endpoint of their maturation are abnormal as to shape, and consequently as to their osmotic and mechanical fragility. They are trapped and destroyed in the spleen far more readily than normal red cells, and the degree of anemia that comes about thereby is conditioned by the ability of the bone marrow to compensate for the unusual loss, most marked during so-called crises when the marrow undergoes temporary depression by virtue of intercurrent infection or some other extraneous influence. This is simply an exaggeration of normal splenic function, and is included in the category of *secondary hypersplenism.*

The second form of hypersplenism which has been firmly established is that due to *production of autohemagglutinins*, which may

be selective for the erythrocyte, the neutrophilic leukocyte or the thrombocyte (platelet), to any combination or to all three. Under these circumstances the spleen plays a dual role. First, it is frequently a major depot for the production of antibodies that agglutinate cells of the blood or damage their membrane. Second, the spleen filters the clumped or otherwise damaged cells from the blood by virtue of its function of *sequestration*.

Less well understood is the possible humoral, perhaps hormonal, control of the bone marrow. Dameshek has suggested that platelet production in idiopathic thrombocytopenic purpura is inhibited by this means, despite the excessive numbers of megakaryocytes. This I doubt, because in a significant number of instances it is possible to demonstrate thrombagglutinins. Nonetheless, there has been the occasional case of marrow depression where removal of the spleen has been followed by regeneration of hematopoietic tissue, implying a splenogenous damper to blood formation.

THE CLASSIFICATION OF HYPERSPLENISM

Hypersplenic syndromes are divisible into two major groups, primary and secondary. Those catalogued as *primary* or *idiopathic* have no demonstrable cause, are not associated with concomitant disease that might influence splenic function or blood formation, and are generally characterized by a normal-sized or only slightly enlarged spleen. In the *secondary group* the background disease is usually evident and the spleen significantly enlarged, except in some instances of the allergenic type. Even though their cause is not known beyond the genetic aspects, hereditary spherocytosis and splenomegalic phases of the so-called hemoglobinopathies are listed as secondary, because the spleen plays only a secondary part in blood destruction through its graveyard function.

TABLE 1

CLASSIFICATION OF "HYPERSPLENISM"

PRIMARY
• ACQUIRED HEMOLYTIC ANEMIA (SOME CASES)
• IDIOPATHIC THROMBOCYTOPENIC PURPURA
• IDIOPATHIC SPLENIC NEUTROPENIA
• IDIOPATHIC PANCYTOPENIC "HYPERSPLENISM"
SECONDARY
• DUE TO SEQUESTRATION OF ERYTHROCYTES
▼ CONGENITAL OR ACQUIRED SPHEROCYTOSIS
▼ SPLENOMEGALIC "HEMOGLOBINOPATHIES"
▼ PORTAL HYPERTENSION
▼ OTHER INTRASPLENIC STASIS
• DUE TO ALLERGIC INFLUENCES
• ASSOCIATED WITH INFLAMMATORY STATES
• ASSOCIATED WITH MALIGNANT DISEASES (ESPECIALLY LYMPHOMAS AND LEUKEMIAS)

THE RECOGNITION OF HYPERSPLENISM

While I will outline a number of aids in the diagnosis of the various hypersplenic syndromes, perhaps the key factor is good common horse-sense in evaluating the patient, his blood-picture, and his bone marrow reaction, being certain that sampling of the marrow has been adequate, and that both smears and sectioned units of marrow have been made available for study. Thus, a significant deficit in cellular levels of the blood without demonstrable cause, associated with regenerative activity of precursors of the missing element(s) in the marrow, would point toward the spleen as the offender, especially if it is enlarged. One must qualify such a statement in several respects, however. Chronic granulocytic leukemia in an aleukemic phase may present a diffusely hyperplastic marrow, rich in nucleated red cells and in megakaryocytes. Conversely, splenic influence may bring about hypoplasia of the hematopoietic tissues, even though rarely.

In connection with *hemolytic anemia*, one should expect to find erythropoietic hyperplasia in the bone marrow with a normoblastic type of maturation, reflected in the peripheral blood by reticulocytosis. Products of red cell destruction should be in evidence through an increase in serum bilirubin, urinary and/or fecal urobilinogen output, and

hemosiderin content of macrophages in the marrow sample. Where spherocytosis exists, the osmotic fragility of erythrocytes should be increased, notably in the hereditary form, less frequently in the acquired. Red cells can also be tested for abnormal mechanical fragility. The presence of autohemagglutinins may be demonstrable by the direct or indirect Coombs' technic, testing for cold agglutinins as well as warm, the use of erythrocytes treated with trypsin or papain, and attempts at elution of antibodies from the patient's erythrocytes. One might anticipate a significant reduction in life span of erythrocytes transfused into the patient, measurable by tagging them with radiochromium or by use of the Ashby technic. Abnormal hemoglobins can readily be detected by their differences in electrophoretic mobility, plus chemical identification of fetal hemoglobin.

From these statements it would seem quite simple to secure the diagnosis of hemolytic anemia and catalogue it rather precisely as to type. In practice this is not the case. Too frequently a marrow aspirate diluted with peripheral blood is interpreted as indicating a hypoplastic state, no further effort being made to obtain units for section. Excessive erythropoiesis is not necessarily provocative of reticulocytosis in the peripheral blood as indicated by current technics. Many times we have noted a markedly hyperplastic marrow, many broad flat bluish cells in the peripheral blood film, but very few of the latter in which a skein could be demonstrated by supravital staining with brilliant cresyl blue. A plethora of reticulocytes or their unstained counterparts (and I have seen them reach over 90 per cent of the total of circulating red cells) will completely mask the expected abnormal fragility in hereditary spherocytosis, and inspection of the blood film under such circumstances would be completely misleading. I remember a boy who fit this picture precisely, and who, as part of the regenerative activity of his marrow, presented a hyperleukocytosis with considerable immaturity suggestive of leukemia. Continued study as the crisis passed disclosed the true diagnosis, and splenectomy was followed by clinical cure.

As to serum bilirubin, the level is frequently normal in proven cases of hemolytic anemia, perhaps from a low excretory threshold, and the measurable output of urobilinogen is an unreliable index. The presence of hemagglutinins is significant, their absence meaningless, and, of course, they are never present in hereditary spherocytosis or the hemoglobinopathies unless by rare chance of concomitant disease. The presence of target cells in excessive numbers or the sickling phenomenon in anemic patients may be completely misleading, unless electrophoretic study shows normal hemoglobin combined with fetal type in the former instance, or the abnormal hemoglobin to be dominant in the latter.

The background of *thrombocytopenia* can generally be sorted out on the basis of an adequate study of the bone marrow. A splenogenous background is almost invariably accompanied by excessive numbers of megakaryocytes and their precursors, and a platelet fringe to the more mature forms is of no importance. Of real significance is the normalcy of the nuclei, clear crisp ones indicating that degeneration of the cells has not been brought about by exogenous toxic influences.

Precisely the same conditions may obtain in thrombotic thrombocytopenic purpura, however, and the diagnosis may be completely missed unless one chances on thrombosed vessels in sections of the marrow units. In our two patients we were not so fortunate, and the diagnosis was not made prior to autopsy, splenectomy naturally having failed to bring about a remission. Review of the sections of the two spleens did not disclose thrombotic phenomena in the portions sampled. The almost uniform association of hemolytic anemia with thrombocytopenic purpura in this condition may afford a clue.

With respect to *splenic neutropenia*, here again a regenerative marrow reaction should be seen, with good maturation within the developmental series. Neutropenia induced by drugs may show hypoplasia of granulocyte

progenitors if the marrow is sampled early, while a specimen in the regenerative phase may be so full of myeloblasts and promyelocytes as to simulate acute granulocytic leukemia. In contrast, chronic leukopenic granulocytic leukemia may resemble splenic neutropenia rather closely.

It is obvious, then, that the recognition of a hypersplenic syndrome is sometimes easy, but more frequently requires the sixth sense that we so often wish we possessed. This will become even more evident when we reach the group of malignant disorders complicated by hypersplenism. For example, one of our patients had a clearly defined hemolytic anemia, and his marrow showed predominantly hemolytic activity. He did not improve on conservative management, and splenectomy was performed six months later. Histologic examination of the spleen revealed the picture of granulocytic leukemia, and this was confirmed by subsequent marrow study. This, then, was one of the uncommon instances in which granulocytic leukemia has been ushered in by a hemolytic phase, during which the basic disease cannot be recognized as such. Still other conditions, such as disseminated lupus erythematosus may bring about conspicuous hemolytic or thrombocytopenic episodes while diagnostic features of the lupus *per se* remain hidden for a time, and unsuccessful splenectomy has been performed occasionally.

THE BEHAVIOR AND MANAGEMENT OF HYPERSPLENISM

One cannot predict with any degree of assurance the results that may be obtained by one or another form of management in a given patient. Left completely to themselves, the vagaries of the various hypersplenic syndromes adopt a wide scatter, from an indolent course with spontaneous remission and relapse to devastating crises with little or no warning. Certain few, notably hereditary spherocytosis and, to a somewhat lesser extent, idiopathic thrombocytopenic purpura, can be issued a clean bill of health, once the spleen is removed without incident and no accessory spleens left behind. Other situations are less predictable, and have given rise to attempts in some quarters toward a more conservative means of management. The only alternate that has been even reasonably successful is treatment with adrenal corticosteroids or pituitary corticotropin, each with its battery of untoward side-effects over extended periods of therapy. Furthermore, a considerable proportion of patients fail to respond to the hormonal regimen, but obtain a remission after splenectomy. In some instances, the secondary hypersplenism incident to the course of lymphomas and leukemias may be broken by combined corticosteroid and radiotherapy, the latter directed toward the enlarged spleen, perhaps supplemented by cancerocidal agents such as the nitrogen mustards. However, once the diagnosis has been established and a supportive marrow identified, splenectomy has, in our hands, brought about more prompt and permanent remissions, with fewer complications. There can be no question but that extended corticosteroid therapy increases the hazards of the operation proper and the postoperative period.

In considering the behaviour and management of the various forms of hypersplenism, primary and secondary, I will include wherever possible the statistical data of Doctor Charles Doan* and his colleagues, accumulated at the Ohio State University School of Medicine, based on the largest series of hypersplenic syndromes managed by a single medical-surgical team. It comprises 828 patients and covers a consecutive period of 25 years (1931–1956 inclusive), 745 having been subjected to surgical splenectomy. Their experience is more meaningful than ours, except in the field of malignant disease.

Acquired Hemolytic Anemia. All gradients of progression and severity may be found in this group of patients, from the indolent to which the patient adapts until red cell levels reach an extremely low ebb, to the explosive where he is prostrated despite a considerably higher erythrocyte count. In Doan's group

* Personal communication.

59.5 per cent obtained complete relief after splenectomy, 6.7 per cent were significantly improved, while 30.5 per cent showed no significant response. Only 40.9 per cent of patients treated with corticosteroids alone improved, but among the failures splenectomy subsequently brought about an excellent result in more than half, while another third showed improvement. On the other hand, patients who were benefited little or none by splenectomy obtained some degree of help from corticosteroids, even though therapy was often necessarily continuous and associated with unpleasant side-effects.

The presence of demonstrable autohemagglutinins affords no guide toward conservative or radical management. A positive Coombs' test may remain so under either circumstance, or may become negative at a varying rate, dependent on the rate of excretion of preformed antibodies should splenectomy or corticosteroid administration inhibit further formation.

Idiopathic Thrombocytopenic Purpura. Rather mild episodes are frequently noted during childhood, and pediatricians tell me that permanent remissions are brought about by Father Time, perhaps more rapidly by corticosteroids. I am apt to see only the more severe cases that are brought to the hospital, and must admit that even a few of these undergo remission without losing their spleens. Nonetheless, I have seen others that were followed as long as four years in a risky thrombocytopenic status before submitting to curative splenectomy. Doan reports an interesting series of 13 pregnant women who developed ITP, 6 of whom had their spleens removed as an emergency measure without interruption of pregnancy. All of the babies lived, but 3 were born with congenital thrombocytopenic purpura, 2 recovering spontaneously and one requiring splenectomy 4 weeks after birth.

Among adults approximately 90 per cent can be expected to be cured by removal of their spleens, while only about 25 per cent obtain satisfactory remissions through conservative management, and some of these require continuous corticosteroid therapy to maintain them. In my opinion, the latter is a waste of time and money, as well as being a continual worry both to patient and physician. For example, one of our patients had her spleen removed and was home a week later with a clear skin and a normal platelet count. A similar patient was treated with corticosteroids and ceased to bleed, but her platelet count failed to rise over a two-week period in the hospital. Splenectomy was followed by a return of platelet levels to normal, but convalescence was protracted. Take your choice.

Idiopathic Splenic Neutropenia. We have only encountered one such patient, and she persists in holding onto her spleen. A therapeutic trial of prednisolone was followed by restoration of circulating neutrophils to low norm, but the count tapered off as the hormone was withdrawn. Her marrow showed compensatory hyperplasia of granulocytic progenitors. Doan has had 25 cases, splenectomy having been performed in all, with only one failure in obtaining permanent remission.

Idiopathic Splenic Pancytopenia. This is also an uncommon syndrome, the depression in all formed elements of the blood being matched by a mass hyperplasia of the marrow. Most of our patients have had an immediate hematologic and clinical response after removal of the spleen, very few failing to obtain benefit, although there have been some instances of recurring cytopenia. One had a decided upswing in blood cell levels, but died of a resistant staphylococcic pneumonia during the postoperative period. Another had a very slow response, the blood picture not reaching normalcy for nearly 6 months, even with hormonal help; the levels declined thereafter until death 5 months later, except for a terminal leukemoid reaction of granulocytes. Thorotrast study during life failed to disclose accessory spleens, but 3 cherry-sized ones were found embedded in the tail of the pancreas at the autopsy.

Secondary Hypersplenism Due to Sequestration. Hereditary spherocytosis, where abnormally shaped and unduly fragile red cells are

entrapped and destroyed by the spleen, has already been mentioned, as well as the virtually iron-clad guarantee of clinical cure by splenectomy, with help obtained by no other means. Even asymptomatic subjects should have their spleens removed to avoid unpredictable crises.

Certain cases of acquired hemolytic anemia may develop a considerable degree of spherocytosis, and the sequestration of such cells in the spleen would naturally augment hemagglutinins in producing anemia, providing an additional indication for removal of the organ.

The spherocytosis incident to severe burns, however, has never in my experience created an analogous situation, the anemia being acute and remedied by transfused blood.

Among the so-called hemoglobinopathies, splenomegalic sickle cell anemia and thalassemia, most apt to occur in the young, may obtain improvement in erythrocyte levels after splenectomy by removing a site of sequestration of the abnormal cells. It has been our impression that crises in children with sickle cell anemia have occurred less frequently after their large spleens have been removed.

Portal hypertension creates stasis within the spleen and leads to the sometimes rather marked enlargement of the organ. Cirrhosis of the liver is the most common cause, thrombosis of the splenic vein a rare one. On several occasions I have made careful dissection of the hilar vessels of large spleens removed under the diagnosis of Banti's syndrome where the liver and splenic vein were quite normal, and demonstrated arterio-venous anastomoses which explained the intrasplenic hypertension and consequent splenomegaly. Under these various circumstances, a moderate panhematocytopenia is apt to develop, and the status of the patient may be appreciably bettered after splenectomy.

Other instances of stagnation of blood in the spleen may be due purely to its great size. For example, the huge spleen of Gaucher's disease can bring about a reduction in level of blood cells of considerable degree, and its removal can restore both blood levels and comfort to the patient. Why, then, should we sit on a big spleen, whatever be the cause?

Secondary Hypersplenism Due to Allergic Influence. We could spend the evening on this topic alone. Suffice it to say that the spleen can be an intermediary between an allergen and blood destruction, and that its removal will virtually always break the destructive cycle. I remember a woman, for example, whose hypersensitivities were so extreme that she went into shock during a standard skin-testing procedure. She had hemolytic jaundice, thrombocytopenic purpura, and marked neutropenia. She was urged to have her enlarged spleen removed, but chose the alternate course of corticosteroid therapy and showed great improvement for nearly a year, despite her becoming hairy, moon-faced, edematous, and hypertensive. Nonetheless, serious panhematocytopenia recurred in the face of continuous treatment. Her spleen was then removed without incident, and a normal hematologic state was restored during gradual withdrawal of the hormone. One can generalize that splenectomy affords as good a result as elimination of the allergen, if it can be identified, and more lasting than treatment with corticosteroids.

Secondary Hypersplenism Associated with Inflammatory States. This is likewise a broad field on which we can barely touch. Examples are the splenomegaly and panhematocytopenia of various gradients associated with rheumatoid arthritis, sarcoidosis, tuberculosis, kala azar, rarely syphilis and malaria nowadays, and so on. A spectacular instance was an 18-year-old colored girl admitted to the Presbyterian Hospital virtually moribund. She was extensively ecchymotic and bleeding profusely from all orifices. Anemia was profound, and no platelets could be found in her blood film, leukocytes being very sparsely distributed and practically all lymphocytic. A marrow aspirate was performed immediately, the smears showing an abundance of all three developmental series, and splenectomy was performed as an emergency procedure the same afternoon. The enlarged spleen was

riddled with fibro-caseous tuberculomata. Several large lymph nodes removed at the same time were completely caseous. Bleeding ceased promptly, but normal blood levels were restored rather slowly. Antimicrobial therapy was instituted, and the girl is alive and well today, eight years later.

Again one can generalize that, if cytopenias are a problem and cannot be improved by treatment of the basic disease, the enlarged spleen should be removed and a good result anticipated. Exceptions should be made in subacute bacterial endocarditis, and in a self-limited disease such as infectious mononucleosis, which is sometimes complicated by hemolytic anemia and thrombocytopenic purpura. Some authors also make an exception of kala azar, but I have seen salvage by splenectomy in this condition.

Secondary Hypersplenism Associated with Malignant Disease. Certain complications arise during the course of lymphomas, chronic leukemias and erythremia that are incompatible with the life or well-being of the patient, how-

TABLE 2

INDICATIONS FOR SPLENECTOMY IN MALIGNANT DISEASE OF THE RETICULO-ENDOTHELIAL SYSTEM

		ANEMIA	HYPERSPLENISM THROMBO-CYTOPENIA	HYPERSPLENISM GRANULO-CYTOPENIA	MASS OR PAIN WITH HYPER-SPLENISM	ALONE	(●) NONE
LYMPHOMATOUS DISORDERS	34	25	18	16	8	4	(4)
FOLLICULAR LYMPHOMA	4	3	3	3	3		
LYMPHOCYTIC LYMPHOMA	5	4	3	3	1	1	
HODGKIN'S LYMPHOMA	8	7	5	1		1	
RETICULUM CELL LYMPHOMA	5	2	1	1	1	2	(1)
CHRONIC LYMPHOCYTIC LEUKEMIA	12	9	6	8	2		(3)
MYELOPROLIFERATIVE DISORDERS	25	18	11	5	7	4	(2)
CHRONIC GRANULOCYTIC LEUKEMIA	10	9	4	3	2	1	
MYELOFIBROSIS	9	6	5	2	3	2	
ERYTHREMIA	6	3	2		2	1	(2)
TOTAL	59	43	29	21	15	8	(6)

(●) THESE WERE POORLY SELECTED CASES (2 FROM PRESBYTERIAN HOSPITAL) WHICH WERE INCLUDED IN THE SERIES TO EMPHASIZE THE NEED FOR CRITICAL EVALUATION OF EACH PATIENT BEING CONSIDERED FOR SPLENECTOMY.

TABLE 3

INCIDENCE OF HYPERSPLENIC CYTOPENIAS

	ANEMIA ONLY	ANEMIA + THROMBO-CYTOPENIA	ANEMIA + GRANULO-CYTOPENIA	PAN-CYTOPENIA
LYMPHOMATOUS DISORDERS	3	6	1	10
FOLLICULAR LYMPHOMA		1		1
LYMPHOCYTIC LYMPHOMA		1	1	1
HODGKIN'S LYMPHOMA	2	4		1
RETICULUM CELL LYMPHOMA				1
CHRONIC LYMPHOCYTIC LEUKEMIA	1			6
MYELOPROLIFERATIVE DISORDERS	5	3		4
CHRONIC GRANULOCYTIC LEUKEMIA	4	1		2
MYELOFIBROSIS	1	1		2
ERTHREMIA		1		
TOTAL	8	9	1	14

ever well controlled the basic disease by established methods. One of the most troublesome is splenic dysfunction as related to blood destruction, less often splenic size and impaired circulation. As to the serious cytopenias that may develop, it is all very well to shrug the situation off as the inevitable time when "the white popsicles eat up the red popsicles and have platelets for dessert," but in properly selected cases the patients may be returned to a comfortable and useful life following splenectomy. Furthermore, the cytopenic states may completely block treatment of the underlying disease except for corticosteroids, but restoration of blood levels has in our experience permitted resumption of other forms of therapy.

Our series is made up of 59 cases, 27 of which were managed at the Presbyterian Hospital, 23 at the Hospital of the University

TABLE 4

CLINICAL AND HEMATOLOGICAL RESULTS OF SPLENECTOMY

	EXCELLENT		MODERATE OR TEMPORARY BENEFIT		NO BENEFIT	TOTAL
	LIVING	DEAD	LIVING	DEAD	DEAD	
LYMPHOMATOUS DISORDERS	14	6		10	(4)	34
FOLLICULAR LYMPHOMA	2			2		4
LYMPHOCYTIC LYMPHOMA	4			1		5
HODGKIN'S LYMPHOMA	3	2		3		8
RETICULUM CELL LYMPHOMA	2	1		1	(1)	5
CHRONIC LYMPHOCYTIC LEUKEMIA	3	3		3	(3)	12
MYELOPROLIFERATIVE DISORDERS	9	4	3	6	1 (2)	25
CHRONIC GRANULOCYTIC LEUKEMIA	4	1	2	3		10
MYELOFIBROSIS	4	3	1	1		9
ERTHREMIA	1			2	1 (2)	6
TOTAL	23	10	3	16	1 (6)	59

PARENTHESIZED CASES WERE POORLY SELECTED. THERE HAVING BEEN NO REAL INDICATION FOR SPLENECTOMY (2 PRESBYTERIAN HOSPITAL)

TABLE 5

POSTOPERATIVE IMPROVEMENT IN BLOOD LEVELS

	ERYTHROCYTES	THROMBOCYTES	GRANULOCYTES
LYMPHOMATOUS DISORDERS	25	18	16
WITHIN 10 DAYS	19	13	12
WITHIN 1 MONTH	4	3	3
WITHIN 3 MONTHS	1	1	1
OVER 3 MONTHS		1	
NO IMPROVEMENT	1		
MYELOPROLIFERATIVE DISORDERS	18	11	5
WITHIN 10 DAYS	15	8	5
WITHIN 1 MONTH	3	2	
WITHIN 3 MONTHS			
OVER 3 MONTHS		1	
NO IMPROVEMENT			

of Pennsylvania, and the remainder were patients about whom I had been consulted in other institutions. This does not lend to uniformity of study or management, but offers at least an average cross-section of what might be anticipated among patients at large.

Indications for splenectomy under these circumstances are shown in Table 2, with hypersplenic cytopenias predominating, while the splenic mass or discomfort provided another indication, alone or in connection with a blood cell deficit. Six cases were poorly selected for the operation, two of them ours. The tabulation also discloses a predominant incidence among the lymphomatous disorders, especially chronic lymphocytic leukemia. It likewise shows that the cytopenias were generally multiple. A breakdown of the latter in individual patients is presented in Table 3, panhematocytopenia being the most common.

The results are listed in a general way in Table 4, being classed as excellent in 33 cases, while 19 more patients obtained moderate or temporary benefit. Only 7 obtained no relief,

TABLE 6

RECURRENT CYTOPENIAS IN 16 CASES AFTER REMISSION INDUCED BY SPLENECTOMY

	NO. OF CASES	PROGRESSION OF DISEASE	CHEMO-THERAPY	RADIO-THERAPY	INFECTION	ACCESSORY SPLEENS	SYSTEMIC ANTIBODY PRODUCTION
LYMPHOMATOUS DISORDERS	11	7	6	2	1 ?	2	
FOLLICULAR LYMPHOMA	2	2	1				
LYMPHOCYTIC LYMPHOMA							
HODGKIN'S LYMPHOMA	4		3		1 ?		
RETICULUM CELL LYMPHOMA	2	2	1	1			
CHRONIC LYMPHOCYTIC LEUKEMIA	3	3	1	1		2	
MYELOPROLIFERATIVE DISORDERS	5	4				1	1 ?
CHRONIC GRANULOCYTIC LEUKEMIA	4	3					1 ?
MYELOFIBROSIS	1	1				1	
ERYTHREMIA							
TOTAL	16	11	6	2	1 ?	3	1 ?

* PERIODS OF REMISSION FROM HYPERSPLENIC CYTOPENIAS RANGED FROM 2 TO 50 MONTHS (AV. 11 MO.) IN LYMPHOMATOUS GROUP AND 1 TO 36 MONTHS (AV. 13 MO.) IN MYELOPROLIFERATIVE GROUP

TABLE 7

INFLUENCE OF AGE ON RESULTS OF SPLENECTOMY

AGE BY DECADES	10–20	20–30	30–40	40–50	50–60	60–70	70–80
LYMPHOMATOUS DISORDERS:							
FOLLICULAR LYMPHOMA			★		○	★ ○	★
LYMPHOCYTIC LYMPHOMA				★	★	★ ○	★
HODGKIN'S LYMPHOMA		★	★★ ○	○○	★★		
RETICULUM CELL LYMPHOMA				★★		■	★ ○
CHRONIC LYMPHOCYTIC LEUKEMIA			★	★ ○	★★★ ○ ■	○○ ■	■
MYELOPROLIFERATIVE DISORDERS							
CHRONIC GRANULOCYTIC LEUKEMIA	○			★	★★ ○○○	★★ ○	
MYELOFIBROSIS				★★	★ ○	★★ ○	★★
ERYTHREMIA				■	○ ■	○ ●	★
TOTAL	○ 1	★ 1	★ 4 ○ 1	★7 ○3 ■1	★9 ○7 ■2	★6 ■2 ○7 ●1	★6 ○1 ■1

★ EXCELLENT RESULT
○ MODERATE OR TEMPORARY BENEFIT
● DEATH DUE TO UNAVOIDABLE COMPLICATIONS DURING IMMEDIATE POSTOPERATIVE PERIOD
■ DEATH DUE TO IMPROPERLY SELECTED PATIENT FOR SPLENECTOMY

and 6 of these should not have had their spleens removed.

Restoration of blood cell levels was exceedingly variable (Table 5), but most patients were greatly improved within 10 days after operation. One patient with profound thrombocytopenia was virtually given up, being carried on corticosteroids for about 5 months, when suddenly the platelet level rose to normal for no good reason and has remained so. Recurrence of cytopenias was noted in 16 patients, the probable causes being listed in Table 6.

An interesting and important observation has been made in the group of 5 patients in which a lymphoma was apparently primary in the spleen. In 3 there has been no evidence of recurrent tumor after 35, 56, and 67 months. One has had nodal recurrence after 24 months, which has responded nicely to radiotherapy. The fifth had a remission for 12 months, but died 20 months later from systemic lymphomatosis.

Among patients that have expired, the range of survival since splenectomy is 4 to 51 months (av. 17) in lymphomatous disorders, and 2 to 42 months (av. 15) in myeloproliferative disorders. Those still living range in the former category from 12 to 54 months (av. 29), and in the latter 2 to 72 months (av. 24).

Age does not seem to be any great hazard with respect to splenectomy in malignant disease, as shown in Table 7, excellent results being richly dispersed among the sixth, seventh, and eighth decades. Furthermore, our group of patients has not displayed undue susceptibility to infection, an observation also made by Doan, et al., in contrast to the views of certain other authors.

SUMMARY

The probable mechanisms whereby splenic dysfunction may bring about reduction in levels of the formed elements of the blood are discussed, as well as the means by which they may be detected. The various forms of so-called hypersplenism are considered briefly, including the indications for splenectomy and the results to be anticipated following the operation. Particular attention is given to the salvage of selected patients with malignant disorders of the reticulo-endothelial system by splenectomy.

Vasospasm: Its Important Bearing on Certain Problems of Hemodynamics[1]

By HIRAM E. ESSEX, PH.D.[2]

IN our early experience with the physiologic effects of a variety of toxic substances my colleagues and I attributed to peripheral vasodilatation the precipitous decrease in blood pressure following their intravenous administration—provided, of course, that the profound hypotension could not be accounted for on the basis of some other demonstrable mechanism. This particular explanation possessed great appeal, perhaps because of its simplicity; but more impelling, I suspect, was the fact that logic could be marshalled in its support. Parenthetically, I might add that someone has referred to the use of logic in such a connection as a method by which one can go wrong with confidence. Further experimentation gradually illumined the darkness and an explanation of the mechanism responsible for the hypotension in question was evolved which, in my opinion, is a much closer approximation of the truth of the matter. The evidence obtained in my own and other laboratories and the bearing of these data on the general concepts of vascular spasm constitute the material I wish to offer for your consideration in this lecture.

PERTINENT INVESTIGATIONS BY OTHERS

Even a superficial examination of the appropriate literature impresses one with the considerable number of papers concerned with some aspect of the important and interesting problem of vascular spasm as a primary consideration in a rather wide variety of diseases and conditions from trauma of extremities to cerebral vascular disorders (1).

Ideas may differ as to how "vascular spasm" or "vasospasm" should be defined. In the opinion of Pickering (2) it is a degree of contraction that is beyond the range of the normal; and when the contraction is sufficient to occlude a vessel and produce symptoms of ischemia in the tissues the vessel supplies, the contraction is pathologic. Floyd and Van Meter (3) have defined vasospasm as an unnatural and abnormal contracture of muscle cells of arteries and veins. Those with a leaning toward teleology emphasize that in the absence of the ability of vessels to contract vigorously—that is, to go into spasm—severe wounds of the larger arteries would more frequently result in the exsanguination of the individual. It indeed could be contended that vascular spasm is an overfunctioning of an entirely normal mechanism which is potentially protective in nature. But given sufficient, appropriate stimulus it may respond with such intensity as to result in occlusive spasm of certain vessels and death of the tissues supplied if not, as is sometimes the case, death of the whole organism.

Many workers have attempted to reproduce in experimental animals the occlusive spasm seen in the large arteries of human extremities. Alexander and Kantrowitz (4) stimulated nerves of dogs by direct and reflex routes, and traumatized femoral arteries by various means, but failed to produce a reaction comparable to the extreme and extensive spasm that occurs in man as the result of many forms of trauma such as fractures of the limbs, gunshot wounds, crushing injury, and the like. On the other hand Simmons (5) recently has reported successful production of

[1] Nathan Lewis Hatfield Lecture XLII, College of Physicians of Philadelphia, 8 January 1958.

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vasospasm in dogs by lengthening the femur of one leg enough to stretch its vascular system. His positive results mark an approach that should yield additional and valuable information on this rather obscure vascular phenomenon.

Kinmonth, Hadfield, Connolly, Lee, and Amoroso (6), however, found it possible to induce by trauma complete spasm in the femoral and brachial arteries of monkeys. Frequently knife dissection and blunt dissection in freeing a vessel were sufficiently irritating to make it contract into a white cord with closed lumen; but when this means failed, rubbing with dry gauze and stretching the vessel were resorted to. With these technics full spasm was produced in 16 of the 30 arteries observed, partial spasm in eight, and none in six. An explanation of the variability in the response of the vessels was not discovered. The negative results obtained by some investigators using similar methods may well be attributed to the species of animal used. There may be a striking difference in the reaction to similar stimuli of the vessels of the monkey compared with that in the dog. In an attempt to discover the cause of vasospasm by study of reactions in the major arteries of the extremities of cats and dogs, Reid (7) applied directly or injected intramurally serum and also defibrinated blood. The results were negative, but arteries which had failed to go into spasm on the application of serum did so when stimulated mechanically.

It should not be inferred that blood vessels become spastic from severe trauma alone, since spasm may occur without the exciting agent or stimulus being discovered. For example, Bay (8) has described localized transient spasms of vessels of one or more extremities of three patients. There were accompanying cold and pain in the affected part which was sometimes incapacitating, but a cause for the spasm was not found. Also, a case of simple spasm of the femoral artery in the absence of obvious trauma has been reported by Hanna (9).

It should not be concluded that vasospasm is an affliction to which the vessels of the limbs alone are subject. Whether spasm of intracranial arteries may be responsible for cerebral stroke and related involvements of the brain is still an unsettled problem. In the opinion of Denny-Brown (10) the chief and perhaps only factor in the regulation of cerebral blood flow is cerebral metabolism. On the other hand, Ecker and Riemenschneider (11) have demonstrated to their satisfaction by a series of angiograms that spasm of intracranial vessels occurs in a small percentage of cases of cerebral vascular disorders.

Spritzler, Corday, Bergman, and Prinzmetal (12) have reported the successful production of spasm of the coronary arteries of the dog, with the suggestion that their results lend support to those who believe that the coronary arteries of man may go into spasm.

Lewis (13) has shown that ergotoxine given into the breast muscles of hens causes constriction or spasm of the arteries of the comb. The constriction is unrelieved by relaxing vasomotor tone or by local warming. The gangrene which follows is not a direct result of the vasoconstriction but is owing to changes induced in the arteries which eventually cause complete arrest of blood flow and death of the tissues.

In the discussion thus far it is obvious that I have not covered all of the available literature on the subject of vascular spasm; nor was it my intention to do so. The chief purpose of introducing the above-mentioned investigations into this discussion has been to provide an orientation for the presentation of the results of pertinent researches with which I myself have been intimately associated.

The results of work which I shall now discuss have, it seems to me, an important bearing on certain problems of hemodynamics.

I shall first present briefly some of the findings in experiments with the extracts of intestinal parasites.

PRODUCTION OF VASCULAR SPASM

Extracts of Parasitic Nematodes. In dogs.—Many investigations have been made with

both crude and refined extracts of Ascaris suum, nematode of swine which is indistinguishable morphologically from A. lumbricoides, the round worm of man (14, 15). A crude extract may be obtained by fragmenting the worms by any convenient method in an equal quantity, by weight, of saline solution. After centrifuging, the supernatant is collected and preserved by adding merthiolate or it may be sterilized by boiling. Frequently 1 ml. of this extract per kilogram of body weight given intravenously to dogs is lethal. When such a dose of the extract is given to an unanesthetized dog a series of very grave symptoms ensue. The animal retches, vomits, urinates, defecates, and becomes prostrate in a few minutes. Death usually follows 10 to 20 minutes later. The most striking finding at necropsy is an intense congestion of the liver and the abdominal viscera.

An intravenous injection of the same dose given to an anesthetized dog causes profound shock, as indicated by a sudden, severe decrease in arterial blood pressure and an increase in the portal venous pressure, with little change of pressure in the posterior vena cava. In addition, the drawn blood does not coagulate. The recipient dies in a few minutes. Should the animal survive the first dose, a second dose of the same size is without much if any effect. If double the initial dose is given after recovery of the blood pressure, a second marked decrease of arterial blood pressure often occurs. With repeated small doses, the animal becomes unresponsive to the extract (tachyphylaxis) and will remain so for many days or even weeks.

Markowitz, Mann, and I in 1931 obtained results almost identical to these in experiments with extracts of the canine ascarid, Toxocara canis (16). Years later, in 1947, with Graña and Mann (17) I did further work on extracts of Ascaris suum and hydatid cyst fluid. Since the liver appeared to be the principal canine organ affected we made a series of studies of the effect of ascaris extract on the liverless dog. To our surprise the liverless animal tolerated with ease doses of the extract that were lethal for a dog with the liver present. When the isolated liver of the dog was perfused through the portal vein with defibrinated blood, an injection of ascaris extract into the portal vein caused a sharp reduction and sometimes a complete arrest of blood flow through the liver, with acute congestion as was seen in livers in situ.

Many other observations made were not pertinent to this discussion, so I shall proceed to the next series of experiments, done with Thomas (18), in which ascaris extract was used.

Since previous work had given ample proof that the liver was seriously affected by injections of the extract, we desired to determine just where in the hepatic circulation the site of action was. Attempts to observe the behavior of the blood vessels by a technic of transillumination were not successful. Then, using vinyl acetate, we made casts of the circulation of the liver of dogs. By this means it was revealed that the congestion of the liver and likewise the increased portal venous pressure were results of spasm of the hepatic veins. The casts of their finer branches had the appearance of corkscrews, reflecting the spiral arrangement of their musculature. At once it became obvious that the hypotension in the dog following injections of ascaris extract was owing to inability of the blood to pass through the hepatic veins, which resulted in pooling of the blood in the liver and other viscera drained by the portal venous system and decrease of the cardiac output. Recently Weil, MacLean, Spink, and Visscher (19), after working with endotoxins of certain gram-negative bacteria, have reported strikingly similar results following intravenous injection of endotoxins into dogs. Although they did not make casts of the hepatic veins of the livers of dogs killed by endotoxins, their data point to partial or complete spasm of these vessels.

Those familiar with anaphylaxis in the dog will recognize that the reactions just described are characteristic of anaphylaxis in that animal. Hence the reason is clear for describing such substances as ascaris extract

and endotoxins as "anaphylactoid." Large doses of histamine also produce results in the dog that are indistinguishable from those that have just been reviewed, but the problem of whether we are dealing with a mechanism in which histamine plays a consistent role must await further investigation. The fact that similar responses can be excited by several agents as widely different as ascaris extract and bacterial endotoxins does not constitute proof of a single initiating or underlying cause for the responses seen.

In rabbits and cats.—Although intravenous doses of ascaris extract when given to rabbits and cats caused a decrease of the blood pressure to a shock level, the congestion of the liver seen in the dog never occurred in the rabbit but occasionally was observed in the cat. Casts of the hepatic vasculature did not show the extreme contracted state of the hepatic veins observed in the dog. As will be shown later with another agent, in the rabbit and cat the circulation in the lungs was more vulnerable than that in the liver. These animals responded to ascaris extract by spasm of the pulmonary arterial system with exception of the main trunks.

D-L Substance in Rabbits, Guinea Pigs, Cats, and Dogs.—Delezenne and Ledebt (20) discovered that 40 per cent egg yolk when mixed with venom of the cobra and the daboia and incubated at 37° C. for 30 minutes produced hemolytic lysolecithin by an enzymatic process. They found also that aside from these lytic properties the mixture acquired extreme toxicity for rabbits and guinea pigs, which responded to an intravenous injection of this mixture in a manner strongly resembling anaphylactic shock. They did not pursue the problem further. Boquet, Dworetzky, and I (21) set ourselves the task of determining the mechanism by which this mixture—or witch's brew if you prefer—which we have called the D-L substance, caused death. It should be stated here that we made the D-L substance with the venom of seven other species of poisonous snakes in addition to the cobra. To eliminate completely the effect of the venom in the mixture it can be neutralized by antivenom following incubation and before injection. The amount of venom added is usually too small to produce a significant response of the circulation of the recipient animal.

For the purpose of the present discussion the remainder of the investigation can be briefly summarized by saying that the mode of action of the D-L substance in the rabbit and cat when lethal was occlusive spasm of the entire pulmonary arterial system except for the main trunks of the pulmonary arteries, and that this again was demonstrated by vinyl acetate casts. The effect of a lethal dose of D-L substance on the blood pressure of the cat was reproduced by occluding the pulmonary artery. The decrease of the blood pressure of the rabbit and cat is owing to the inability of the right heart to force the blood through the constricted pulmonary arteries to the left heart, which results in failure of the right heart itself.

The cause of death in the guinea pig appears to be acute bronchospasm as in anaphylactic shock in that animal.

The dog appeared to be more resistant to the D-L substance than the guinea pig, rabbit, or cat. A dose of 1 ml. per kilogram was usually lethal for the rabbit and cat, but the dog recovered from doses of this size. The typical response to an intravenous injection of 1 ml. of D-L substance per kilogram was marked arterial hypotension accompanied by an increase of portal venous pressure. Therefore the D-L substance in the dog produced effects much like those of ascaris extract. Vinyl acetate casts of the hepatic veins show them to be constricted but to a less degree than is produced by ascaris extract.

Protamine Sulfate in Rabbits.—In pursuing another investigation Parkin and I (22) discovered that protamine sulfate (salmine) caused occlusive spasm of the arteries of the ears of rabbits when given intravenously in the proper dosage. Solis and I (23) explored the effect of this agent on the arteries of the ear and other parts of the vascular system.

By use of the Clark-Williams (24) pre-

formed type of transparent chamber, developed at the University of Pennsylvania, direct observations were made with the low power of the microscope of the behavior of the medial artery in response to various doses of protamine sulfate in the unanesthetized animal. Intravenous injection of 20 mg. per kilogram in a series of eight rabbits were followed in five cases by complete disappearance of the lumen of the medial artery; in the other three the degree of constriction was 60, 50, and 33 per cent, respectively, as determined by measurements with an ocular micrometer. Smaller doses of the agent gave negative or inconstant results. The duration of occlusive spasm varied considerably. The lumen usually began to reappear in 2 to 5 minutes. The vessel displayed repeated periods of vasomotion with rhythmic narrowing and dilation of its lumen which returned to the control status usually in 15 to 30 minutes. Similar experiments were performed on rabbits with denervated ears. The injections of protamine resulted in definite vasospasm of the medial arteries which was more intense and lasted longer than in the ears with nerves intact. In one animal in which the occlusive vasoconstriction persisted for more than two hours the ear subsequently became gangrenous.

Results of experiments on isolated, perfused ears of rabbits were in every respect similar to those obtained with the intact ear. During the control period of observation there was a regular and uniform rate of flow. In the first 10 minutes after 15 mg. of protamine sulfate was received into the circulation of the ear, the flow from the ear was reduced 60 per cent, and after 30 minutes' perfusion the reduction was about 30 per cent.

These observations lend strong support to the belief of many investigators that vasospasm may be entirely independent of the influence of the nervous system.

Additional evidence of spasm of the auricular artery was obtained by means of angiography. Angiograms made 1 minute after the injection of protamine showed occlusive spasm of the auricular arteries so complete that practically all evidence of vascularity was absent.

In another series of observations the changes in diameter of the medial artery were correlated with the changes in temperature of the skin of the ear, measured with a thermistor ensemble. The animals were anesthetized with pentobarbital sodium and placed in a box whose temperature and humidity could be kept constant. In all but one of 10 experiments an intravenous injection of 20 mg. of protamine per kg. caused a decrease of as much as 4° C. in the skin temperature of the ear. At the same time, the medial artery had decreased from 1.1 mm. to 0 mm. in diameter. Simultaneously measured changes in the temperature of the skin of the outer surface of the thigh were slight.

The systemic blood pressure of the rabbit decreased following injections of protamine. When first observed this seemed a serious contradiction to the spasm seen in the arteries of the ear, but a series of angiograms of the vessels of the hind limbs showed little change in the arterial circulation following injections of protamine, thus indicating that the arteries of the ear of the rabbit are peculiar in this regard.

It was observed that in rabbits pulmonary blood pressure too was little changed by protamine.

Myograms of the isolated perfused heart indicated that after administration of protamine a definite reduction in the strength of ventricular contraction lasted for 1 or 2 minutes. The hypotension caused by injections of protamine into rabbits may be owing to the direct effect of the drug on the myocardium or indirectly to the weakening of the cardiac muscle by the temporary reduction of the coronary arterial flow, as this change also was demonstrated in the perfused heart after protamine administration. Whatever may eventually prove to be the proximate cause of the decrease in systemic blood pressure resulting ultimately from protamine, one may confidently expect it not to be peripheral vasodilatation! Attempts to account for the vascular phenomena on the basis of liberation of histamine have failed. Since evidence of even a slight degree of desensitization was not

seen, protamine should not be considered as an anaphylactoid substance.

TREATMENT OF VASCULAR SPASM

At this point will be considered the various means employed to relieve vascular spasm when, unhappily, it has occurred.

It is a common observation that one of the most effective stimuli for increasing the flow of blood to an organ or part of the body is anoxia. Therefore it might be reasoned that the ischemia of the wall of a blood vessel in spasm should normally result in the relaxation of the vessel with automatic re-establishment of the flow of blood through it. But unfortunately blood vessels do not follow a rational pattern or procedure. Cohen (25), in his excellent review of the subject of traumatic vascular spasm, stated that a major artery may remain in spasm for 20 minutes to 24 hours and the spasm may persist until the limb dies. He believed this type of vasospasm not to be traceable to the autonomic nervous system but rather to be a function of the constricting mechanism inherent in the artery itself. In his experience arteriectomy had not relieved traumatic vasospasm, nor had interruption of the sympathetic nervous system.

Similarly, Cranley and Herrmann (26) have expressed the belief that traumatic arterial spasm is caused by direct injury of the artery and treatment should be based on surgical exposure of the vessel for the purpose of discovering the offending agent.

Kinmonth and co-workers (6) have obtained relaxation of traumatic spasm of the arteries of monkeys by keeping the exposed vessels covered as much as possible with a 2.5 per cent solution of papaverine sulfate. The vessels usually responded with noticeable dilatation in 5 to 15 minutes. The rate of relaxation varied with the size of the vessel and the degree of spasm. The investigators also have obtained relief of vasospasm in 13 among 14 cases of spasm of arteries of man by local application of papaverine.

Edwards and Lyons (27) have recommended the application of warm saline, procaine, or papaverine solution to the exposed vessel in cases of traumatic arterial spasm. If contusion or thrombosis of the artery is present they advise resection of the injured area and replacement with a graft.

Smith and Rees (28) used continuous spinal anesthesia with procaine for as long as 69 hours and 45 minutes to relieve vasospasm and pain in cases of peripheral embolism. They advocated surgical intervention if improvement of the condition were questionable following sympathetic block.

For relief of vasospasm produced reflexly by cold, Green (29) has reported satisfactory vasodilatation of vessels of the tips of the fingers and toes and of the forehead in human subjects by use of adrenergic blocking agents, tolazoline (priscoline) and azapetine (ilidar). The drugs were administered intravenously over a period of 30 to 45 minutes.

For vascular spasm which is the result of stretching of the vessels incident to correcting flexion deformities or to returning the extremity to normal length after a considerable period of shortening, Simmons (5) has advocated relief of the stretch on the vessels as soon as possible after discovery of the spasm.

Weil, Hinshaw, Visscher, Spink, and MacLean (30) have reported that intravenous injection of the vasopressor drug metaraminol (aramine) counteracted the mechanism responsible for the pooling of blood in the abdominal viscera of dogs caused by injections of endotoxins. Whether this drug is of value in dealing with the spasm of the hepatic veins caused by ascaris extract has not been determined.

In the work with Graña and Mann (17) already referred to, premedication with 6 mg. per kilogram of diphenhydramine (benadryl) was ineffective in preventing the vasospasm of the hepatic venous system caused by ascaris extract in the dog. Recently Peters and I have had more promising results with chlorpheniramine (chlor-trimeton), but all attempts to relieve already existent hepatic venous spasm by antihistaminics or relaxing drugs given intravenously have been without apparent success. The solution of the problem of getting the drug into contact with the tissues

of the vessel in spasm is not apparent. Direct application of the drug to the vessel as is done with the vessels of the extremities is not a practical procedure when one is dealing with visceral blood vessels such as hepatic veins and pulmonary arteries.

COMMENT

I hope that I have not taxed the patience of my audience in my efforts to bring together in a single presentation the various aspects of the phenomena of vasospasm. The possibility that vasospasm may be an important factor in the etiology of many obscure conditions should not be ignored. As Weber (31) observed nearly a decade ago, "Many visceral phenomena may be of hemangiospastic origin, including the rare cases of symmetrical necrosis of the renal cortex (especially in puerperal women)." However that may be, I shall be gratified if this discussion stimulates thinking in such a direction.

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Problems and Trends in Medical Education[1]

By ROBERT A. MOORE, M.D.[2]

THE needs and demands of society change continuously. In any short period it is not always possible to see this change, but if we compare the situation 10, or 20, or 50 years ago with that of today, there are striking differences.

I am reminded of a statement made by one of America's great medical scientists, Dr. Peyton Rous, some years ago when he presented his first report on the Shope papilloma of rabbits—almost 30 years after he had studied the Rous sarcoma of chickens. He said, "Science is like a snake. It crawls along on its belly slowly and continuously and only rarely rises up and strikes."

As an example, 50 years ago Mr. Abraham Flexner was engaged in his now-famous survey of medical education which brought to public view the sad state of this form of professional education. Think of what has happened since then: the proprietary school no longer exists; colleges of medicine have increasingly become a part of universities; didactic teaching has given way to laboratory and clerkship experiences; faculties composed only of local practitioners have had added a core of full-time members, and medical research has assumed a large role in the activities of faculty and students. Or, let us view the situation just 20 years ago—in 1938. Penicillin as an effective agent in infection had not been discovered to usher in the era of antibiotics. Mental health as something more than a taboo to be hidden in institutions was just emerging and colleges of medicine here and there were giving consideration to establishment of departments of psychiatry and expansion of instruction in this area. The American Cancer Society had not yet evolved out of its predecessor and the budget of the National Cancer Institute was reckoned in a few hundred thousand dollars rather than the almost sixty million of this year. The Public Health Service, under the leadership of Dr. Thomas Parran, had not yet taken "the wraps" off the disease syphilis and brought it into the open where it could be controlled. Isotopes were known, but investigators were just beginning to use the heavy isotopes to analyze metabolic processes. The radioactive isotopes were still something for the future.

Even in the ten year period since 1948 there are clearly apparent changes. In the year 1948, 24,434 individuals made 88,244 applications to enter medical school in September 1949. For the class entering in the Fall of 1956 there were 59,798 applications by 15,917 individuals. Each prospective medical student is still applying to about three and a half schools on an average but there were only 65 per cent as many applicants. In the interim new schools have been established—Florida, Miami, Kentucky, Einstein, U.C.L.A., and University of Washington, and two year schools have expanded to four years at Utah, Mississippi, Missouri, and North Carolina, so that in 1956 there were 7,824 freshmen as compared to fewer than 7,000 just a decade before. Actually about 200 more freshmen were admitted in 1956 than in 1955.

Over the half century of the development of modern American medical education since the publication of Bulletin No. 10 of the Carnegie Corporation, or, as it is better known, the Flexner Report, those universities, those colleges, and those groups, which could best see into the future and plan for it have taken leadership and become the leading schools. Johns Hopkins, before the turn of the century, set the pattern that the physician should also be an educated man and required four years of collegiate work of a liberal nature for admis-

[1] Mary Scott Newbold Lecture LXXVI, 5 February 1958.

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sion. In 1917 Washington University and Johns Hopkins established full-time professorships in the clinical departments. In the 1920's the University of Chicago School of Medicine was founded with a totally full-time faculty. More recently, Western Reserve has introduced a new concept of the educational process for medicine. And last year, the cycle was completed when the original experimenter and leader, Johns Hopkins, again seized leadership in announcing a new program for some students of seven years of college, medical school, and internship.

In the thirty years that I have served in medical education, 18 years on the active faculty and 12 in an administrative post in five universities, there have been profound changes. Eighteen years ago, in 1940, almost three-fourths of the time of the junior year and one-third of the senior year at Washington University were lectures, and anatomy occupied 750 hours of the freshman year. In the Fall of 1941 a new curriculum was introduced and today there are few schools with undue orientation to morphology and didactic clinical instruction.

With this background, let us now turn to a crystal ball and see if we can discern some of the problems and trends of medical education in the United States. In doing this, I would like to assume that "I am thinking out loud" and not presenting a formal paper with conclusions and recommendations, such as would be expected in a scientific report.

One of the problems which looms large is the need for graduates of our medical schools in relation both to the demands of medical care and the growth of the population. It has been estimated that to preserve the ratio of one physician to 750 in the population, we will need by 1970 the output of another 31 medical schools, each with a class of 100. When we think there are now only 85 schools in being or in development, the creation of more than a third, again this number assumes a herculean task.

A closely related problem is the projected curve of men and women of college age who will wish to become physicians. The so called "crop" of babies from the 1940's is almost upon the colleges and will soon be on the doorstep of the professional schools. By the late 1960's there will be between 22,000 and 32,000 applicants for medical school as compared to about 16,000 in the Fall of 1956.

How are we to provide more physicians for America and more places for those who wish to be physicians? Manifestly, the easiest solution is to increase the size of each class in each existing school. But, there are limits to such expansion beyond which the quality of the program is lowered to the danger point. I believe that most of the possible expansion of this type has been made in the last ten years, and I suspect in some instances the expansion has already impaired the quality of the educational program.

In my own school, the Downstate Medical Center in Brooklyn of the State University of New York, the freshman class has increased from 100 in 1949 to 150 now, and we are pledged to take 200 within two to three years. Fortunately, the new building for the basic science departments on Clarkson Avenue, has class laboratories for 200 students without crowding, and the State has accepted the principle that there should be one faculty member for each 20 students for each 150 to 250 clock hours of instruction per year. Thus, unless we encounter difficulty in recruitment of faculty, a major problem which I shall discuss later, we have reasonable assurance of the maintenance of quality even with a class of 200 students. There will probably be some loss of intimate contact between senior staff and students which is possible with smaller classes, but we will seek other devices to offset this element of professional education.

A technique to make possible large annual classes but hold the size of each instructional group small is the so called platoon system of admitting and graduating two classes or four classes a year. This has been done at the University of Tennessee for many years. If correlated with other programs and activities it serves the same purpose the "house plan" in a

University does. Four years ago our younger son was in process of applying to college. He set the characteristics as "a men's college of small or modest size in the East." When I encouraged him to look at Harvard he demurred and said it was too large. After a visit there to the houses he changed his mind. Thus by breaking up a large student body into eight or ten smaller groups, each of which eats, lives, and studies together, it is possible to preserve the advantages of the strong faculty and resources of a large university and the advantages of the small university. I do not know when the analog of the house plan should be applied to medical schools, but certainly it is not far above 150 to 200.

Another technique, from which most faculties shy away, is a longer school year. Most medical colleges have a school year between 32 to 36 weeks for four years or 128 to 144 weeks, an average of 136 weeks. If the school year were 44 weeks, still leaving eight weeks vacation each year, the same amount of instructional time would be completed in three years. This is one of the elements in the Hopkins plan. Over a period of 12 years a medical school with 100 in a class would graduate 1600 rather than 1200 under the present schedule. Many, including myself, have raised some objections to this acceleration, largely on the basis of a bad experience during the war, when some of us believed that acceleration had extended to the point where we had students with the viewpoint and attitude of high school students in medical school. If there were careful evaluation of those who are permitted to accelerate in college, as the Hopkins plan envisages, this objection to acceleration in medical school would vanish. It would, of course, be necessary to increase the size of the faculty one third since each department would give its course four times in 12 years instead of three times.

Still another technique is the establishment of new two-year medical schools. The clinical instructional resources and the potential faculty members of some of our present medical schools are greater than the same resources and faculty at the preclinical level. Further, there are many superior universities with strong development and resources in the natural sciences which could form the nucleus of a two year medical school. This has the additional advantage in which the past tells us that two year schools gradually evolve into four year schools.

Thus, as I look in the crystal ball, I see more medical graduates to meet the needs of America, some, in small numbers, from expansion of present schools; some, in large numbers, from acceleration of medical education by a longer school year and by admission of multiple classes in a year; some, in significant numbers, from newly established four-year schools; and some, from coordinated programs between new two-year schools and existing four-year schools. Unfortunately my crystal ball is only qualitative and does not have a built-in electronic computer so I can not tell you how many more graduates there will be, but I suspect the number will not be short of the needs of the American people. May I paraphrase a statement from the financial world: "Don't sell American medical education short."

Let us now turn to the problems and trends of the educational program. I can not agree with those who believe there has been little or no progress and that what we need is a complete overhauling of the pedagogic principles and the curriculum; that is, another 1910. There have been, in my opinion, remarkable improvements and changes in the postwar years. Education is like the research to which Dr. Rous referred and one might say "Education is like a snake. It crawls along on its belly slowly and continuously and only rarely rises up and strikes." Unfortunately many see us only when the snake rises up and strikes, and do not see the slow forward movement. We make the headlines when Western Reserve revises the curriculum, or when Hopkins announces a new program, or when Florida or Stanford establishes a new school fully integrated with the other colleges and schools of the University. We receive no publicity, or, at

the most, a small note on an inside page when we allocate more time to the behavioral sciences, or reschedule the outpatient work so that the program revolves about the patient and student rather than around the faculty member, or when we institute a home care program or assign a family to each freshman student, or when we establish a major clerkship in psychiatry.

We are coming to accept what is a growing characteristic of American society—nothing is worthy of interest and perhaps of support, unless it is spectacular, unless it is new. The snake crawling along on his belly must shift for itself and live on its body stores. Please do not misunderstand me. I want the snake to rise up and strike from time to time. I applaud when it does. I want the snake to be given extra nourishment when it does. But, an animal organism or a society which spends all the time rising and striking will soon wear itself out even with abundant food supply. The way to find the causes and cures of cancer is not to appropriate $100,000,000 in one year and command the scientists to find it, but, rather, to nourish well the snake crawling slowly and surely forward on its belly. It will rise up and strike more certainly under these conditions than if it is constantly stimulated to strike.

But, that is enough of a personal tirade in a public address before a scientific audience interested in medical education. There is not time to review in detail the problems and trends in the medical educational programs, but let me sketch some in broad terms.

The one foremost in my mind is the need for greater emphasis on general liberal education and on the behavioral sciences for the physician in the society of the future. This has perhaps assumed larger proportions in my mind in the four months of the post-sputnik era. Hardly a day passes that someone does not urge that we teach more and better mathematics and science in college, in high school, and even in grammar school, because the Russians are doing so, and look, they have launched a satellite because they did so orient their schools, and we have no satellite in space. Actually, two days before I wrote this, America placed a satellite in the far reaches of space up to 1,700 miles.

Again, please do not misunderstand me. We must do everything necessary to preserve our society, but let's make sure it is our society we are preserving and not someone else's society. I think our method of producing educated men and women is superior to theirs. It has become fashionable to make fun of the three R's and of McGuffey's readers, but they are some of the elements which have made America great. I would not exchange our way of life for theirs for one moment. I think that an engineer who has a background in sociology, and history, and philosophy is a better engineer. And I know that a physician who is aware of the problems of cultural anthropology, and of the relation of health to housing, and to family life, and to economics is a far better physician, than one who is completely oriented to the physical aspects of health and disease.

Do we wish to have a society in which half the population are walking electronic computers and the other half walking laboratories of test tubes and of geiger counters and of cyclotrons? All of this is quite aside from the fact that it takes all kinds of minds to make up a superior and balanced society, and some minds can not assimilate mathematics or physics even if life depended on it. The motto of my own State University of New York is a good one, "Let each become all he is capable of being." This does not mean we will force our own and the next generation into a mold just because someone else has done so.

This leads to another problem and trend to which I give high priority and that is, the importance of basic science. I refer here both to education and to research, and it is closely related to a point which I mentioned earlier, the recruitment of additional academic and research personnel.

The release of energy by fission and fusion was not discovered de novo but came after men and women had studied "truth for truth's sake" about the atom, the interrelations of the

atoms and the internal structure of the atom with a nucleus and electrons. Neither Russia nor the United States could have placed a satellite in orbit without knowledge of the basic properties of metals in response to temperature change, of the properties of liquids and solids and gases and conversion from one phase to another at enormous speeds and many other basic facts. Penicillin was not discovered because a scientist set out to find a treatment for infection, but rather because a bacteriologist was interested in the growth of Staphylococci and had an observant eye and mind. One form of the treatment of coronary occlusion stems from studies by a chemist on the spoilage of ensilage on the farms of the Midwest.

Developmental and applied research and what I call directed or organized research have their place and time, but in our materialistic and industrial society we should not overlook the man of original ideas, the seeker of "truth for truth's sake." This man means as much or more to the next decade and to our survival than those applying the knowledge of the last decade to today. There is an old story about the goose which laid the golden egg. Without a goose there can be no more golden eggs, and not even a goose which lays golden eggs can live and multiply itself without sustenance.

Again, we are caught in that characteristic of our society—a worship of the spectacular and of the practical. The organic chemist studying the basic properties and variants of desoxyribonucleic acid is, in my opinion, just as likely to contribute to the solution of cancer as the experimentalist or clinician studying cancer in animals or man. Modern surgery rests on long years of accumulation of knowledge about neurophysiology, about salt and water balance, about osmotic pressure of the plasma proteins and about the physiology of respiration and circulation, to mention only a few. Here is Dr. Rous' snake again, slowly but surely crawling along on its belly and rising up to strike when Evarts Graham first did a one-stage pneumonectomy or when Alfred Blalock first repaired a congenital anomaly of the heart.

You may be saying: "This speaker is off on a tangent again and has forgotten his topic this evening is, problems and trends in medical education." My answer is: "Not at all. I am directly on the beam." These are the thoughts which those charged with nurturing and administering our universities, our medical colleges, our private foundations, our government agencies, and our voluntary agencies must have if we are to have the universities, and the physicians, and the society we want and need.

It is this kind of thought which has led to introduction of graduate educational programs at the Ph.D. level into our medical schools. It is this kind of thought which has sparked the trend to research by medical students during the summer and during a sabbatical year. It is this kind of thought which has inspired the conversion from a program of spoon-feeding of facts to a program which stimulates the student to think and reason for himself and to project learning rather than cook book experiments. It is this kind of thought which has provoked many to speak out against the support of everything in categories, and largely in applied categories, and hope and wish for support of men with ideas—support of brains and not just of hands. It is this kind of thought which has eventuated in more full-time faculty; in strong departments of psychiatry supported with psychologists, social workers, and cultural anthropologists; in integrated outpatient instruction; and in the many other advances in medical education.

Now, let me turn to the last problem and trend which I wish to discuss. Repeatedly throughout this discussion I have used the words American medical education and American medicine. Actually today it is a great deal more than that. At the turn of the century there was a one way well-worn path between the United States and Europe, particularly to Germany and Austria. After World War I this path was less well traveled and after World War II the direction of travel was reversed. In addition, paths which previously led from the far corners of the Earth to Europe became overgrown from non-use and new paths and

even broad roads were opened up from these same countries to the United States.

It has become clear that the greater part of the World is looking to America for leadership. I hope we have the wisdom and the statesmanship to seize and to hold this leadership. I believe we should and have for at least two reasons—for the sake of humanity, and for our own survival.

The desire for health and happiness does not recognize international borders, color of skin, national origin, or religious belief. Contrarily, the causative agents of disease do not recognize these factors either. The world today is too small for any group in any nation not to help other groups in other nations to attain their just desires for health and happiness. There are many aspects of help and assistance. For the short term there is the loan of public health personnel to bring certain diseases as malaria, dysentery, and typhoid under control. This is being done in many countries through W.H.O. and the United States Overseas Missions. Malaria is no longer a problem in most of the large cities. Far more important is assistance for the long term and this means assistance in medical education, both undergraduate and postgraduate.

It has been my privilege to visit most of the medical schools and the medical care facilities in Southeast Asia in 1952 and again in 1957. The first trip was made for our government and the recent trip was undertaken as a Trustee of the China Medical Board of New York, a private foundation dedicated to assisting with medical education in Asia. Much was accomplished in the five-year interval, but much more remains to be done. Faculty members must be trained both there and in this country In some nations this program is well advanced. For example, in 1957 there were 142 full-time appointed members of the faculty of the Siriraj Medical College in Bangkok. Thirty-two, or a fifth, were on leave for study abroad. In other nations this type of program is less advanced or is presently impossible on any real scale. The medical college at Saigon, which was established by emigrees from Hanoi following the Geneva accord, has not over 30 faculty members, about half of whom are part-time. Only three men are on leave for graduate study in France.

The need for visiting professors is far less at Siriraj than at Saigon. In the latter school a period of years with assistance there by visiting professors must precede or occur concomitantly with training of future faculty. Fortunately the United States Overseas Mission and the American Embassy to Viet-Nam have interested themselves in medicine and are committed to a major effort in this field.

Some of the problems of medical education in these countries have a familiar ring to those we have or have had. Let me cite just a few. There are too many students for the facilities and number of faculty. The full-time faculty are underpaid so they must supplement their income by practice in the late afternoon and evening. Many students come from families with limited financial resources and there are limited scholarship and loan funds. I sometimes wonder if the word frequently used of "underdeveloped countries" is the correct one. It is rather the stage of development and the emphasis. On the other side, there is hardly a school in the Far East which does not have dormitory facilities for half or more of the students.

Finally, if we do not assist these countries in the name of humanity we must do so for our own survival. Let me tell you a story—one I have told many times and perhaps some of you have heard it. In 1952 I was visiting a small rural hospital in one of the nations of Southeast Asia. As the physician-in-chief and I walked through the men's ward, we came to a bed in the corner of the room. On a bed of wood without springs or mattress or linen, other than a thin rattan mat over the wood, was a strapping young man in his twenties. He was sweating and the respirations were shallow and rapid. He did not speak English and I did not speak his native tongue, but his eyes and his face told me more than words in a universal language. They seemed to say, "Please, doctor, let me get well to return to my wife and children." A glance at the chart showed a signifi-

cant continuing elevation of temperature for at least two weeks. A brief discussion with the physician revealed he had typhoid and was entering the third week of the disease. As I started to reach for the chart the physician anticipated the information I sought and said "No, he has not received chloromycetin. We do not have any." I left the hospital a few minutes later and never learned the outcome, but if I know any medicine, it is likely this young man with the appealing eyes died of perforation or hemorrhage.

Also, if I know anything about the methods of our antagonists for a way of life, the wife of this man had a caller a few days later. His pitch, and I use the word advisedly was: "I am sorry your husband died. There is a drug which would have cured him. The decadent war-mongering governments to which your government looks for support did not furnish the drug. If your government had oriented itself to us, we would have provided the drug and your husband would be alive. Will you join with us to overthrow the government?" What would you have answered? Belief in democracy, in self-government, in a way of life are dear, but, the life of a loved one may be dearer.

Many years ago public health officers spoke of an island in a sea of disease. We have gradually enlarged the island. We are not yet an island in a sea of Communism and, in my opinion, the most effective way to hold ourselves as a continent and not to become an island is to show that our way of life is the best. One of the attributes of our way of life is the preservation of health and the prevention of disease.

And, thus, I come to the end of my discussion of "Problems and Trends in Medical Education." If I were to attempt to summarize it would be in these words. American medical education has come a long way since 1910. Let us experiment and improve it further. Let us insure that our future physicians are educated men and women capable of thinking and reasoning. Let us provide the World leadership which is so desperately needed and desired by those looking to us for it. And, last, let us keep our eyes on the ultimate goal of a healthier and happier world, medically, economically, and politically.

Historical Remarks on the Interrelation of Human and Veterinary Medicine[1]

By JOSEPH V. KLAUDER, M.D.[2]

I CAN appropriately begin my presentation with the following quotation from Rudolph Virchow (1821–1902): "Between animal and human medicine there is no dividing line—nor should there be. The object is different, but the experience obtained constitutes the basis of all medicine."

This remark of Virchow some 75 years ago has been paraphrased by a number of writers. A recent rendition of it is by Cameron (1) who writes: "Medicine, of course, is medicine and veterinary medicine is only part of an even greater association of which the most important, largely because of the value of the patient, is human medicine. There is no fundamental difference between human medicine and veterinary medicine. Both apply all the same sciences to different animals and the main distinction lies in economic consideration and in the varying value of the signs and symptoms of the patient."

I, too, may paraphrase Virchow's remark—Medicine is a science concerned with study and disease of all living creatures. There is no dividing line in nature of disease of man and of animals. Human and veterinary medicine are both concerned with the greatest of all mysteries—life and death and equally concerned in the constant battle to conquer disease and its dissemination and vigilance against new diseases. Both professions share the reward and thrill of accomplishment. What is more dramatic than the rising to her feet of a cow in post-parturient collapse after an intravenous injection of calcium? This is equally akin to the spectacular effect of a tracheotomy on a cyanotic child gasping for life.

If there is a dividing line, it would exist in the realm of mental diseases, since psychosis does not occur in animals. Here the dividing line is less than would appear on the surface since neurosis occurs in animals and psychosomatic symptoms; perhaps, too, in the realm of psychiatry belong such symptoms as masturbation of high tension race horses, and behavior problems in animals and fowls. Particular reference is made to "homing" or the direction finding in certain animals—especially dogs, pigeons and migration of birds.

It is interesting to observe the evolution of the realization of Virchow's concept. It appears to me that its realization has been slow, but within recent years there is much evidence of its consummation. I refer to the increasing representation, later discussed, of the veterinarian in the domain of public health. More pertinently, now, to the appearance of textbooks in which "man and animals" appear in the title: For example,—"Some Protozoan Diseases of Man and Animals" (2), "Calcium and Phosphorus Metabolism in Man and Animals with Special Reference to Pregnancy and Lactation" (3), "Comparative Anatomy of the Eye" (4), "Toxoplasmoses of Man and Animals" (5).

In the absence of these words in the title I refer to such textbooks that include animals in the subject discussed; for example, Willis' textbook on "Pathology of Tumors" (6) in the preface of which he writes: "My conviction that much has yet to be learnt from the comparative pathology of tumors is shown by a chapter on this subject. . . ."; the comparative aspects of bacteriology and virology in Topley and Wilson's (7) textbook on bacteriology and

[1] Read before the Section on Medical History, College of Physicians of Philadelphia, 19 November 1957.

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immunity; the chapter on vitamins in animal nutrition in the textbook "Recent Research on Vitamins" (8); the inclusion of some aspects of veterinarian medicine in the analytic review of the bulletin of the Pasteur Institute.

Within the span of my knowledge of medical literature I believe there is increasing evidence of the awareness of comparative aspects of medicine.

The dividing line between human and veterinary medicine is beginning to disappear. The following circumstances doubtless contributed to this change. The evolution of our knowledge of vitamins, hormones, of chemotherapy, and antibiotic therapy focused attention to the common basis in treatment of many diseases of man and of animals; the contributions of veterinary medicine to human medicine; the realization of the important role that veterinary medicine has always played in public health; the increasing number of diseases of animals communicable to men. Modern means of transportation have focused attention to these diseases. The establishment (1948) of a Veterinary Public Health Section of the World Health Organization.

I do not believe knowledge of any disease or pathologic process is complete unless it is known or studied in both man and animals. Many instances of bacillary, viral, mycotic, and parasitic diseases could be cited. This applies to such subjects as pathology, bacteriology, immunology, dermatology, ophthalmology.

No other professions are so closely connected as are those of human medicine and veterinary medicine The desirability becomes apparent of a yet closer active, natural liaison within the framework of some educational scheme.

Mueller (9) advocates a union of veterinary medicine and human medicine and writes of "universal medicine." The latter concerns all living creatures. In such a curriculum basic instruction would be the same for students destined for either human or veterinary medicine. At a certain period in this curriculum the students would be divided into those becoming medical doctors or veterinarians.

Sir Henry Dale (10) likewise discusses this union. He writes: "The problems of pre-clinical education in the two disciplines, medicine and veterinary medicine, are largely a matter of mutual interest and concern. It is at the later stage of direct, so-called clinical education,—whether clinical means literally at the bedside, or in the field when the general physiological and pathologic data and principles acquired in the lecture room and laboratory come to be applied to the recognition, treatment or prevention of disease—that their needs begin to diverge to an important degree, though still with many parallel stretches.

EVOLUTION OF VETERINARY MEDICINE

The conjoint teaching of Veterinary medicine and human medicine is not a new thought. The earliest references to diseases of animals were in conjunction with the first medical writings. Aesculapius in mythologic history includes a knowledge of horses which he derived from Chiron, the Centaur. Hippocrates described the symptoms of diseases and the remedies to be used in animals. Aristotle wrote a book on animal medicine and the Greeks studied and taught both branches of medicine and appreciated to a remarkable degree the dependence of human on veterinary medicine.

References to medicine of man and animals is given in early Hindu, Egyptian and Babylonian literature. According to some writer's (11) interpretation of Vedic records, man acquired the art of medical practice by observing animals and birds and in this sense medical science originated from veterinary medical observation. For example, elephants with colic were observed to eat saline earth. Analogous observation today would concern the eating of the placenta by animals. What symptoms would ensue if by planned experiment the eating of the placenta would be prevented?

In early periods, veterinary medicine was in high esteem and intimately associated with human medicine. At later periods veterinary science lagged far behind human medicine. The ancients called medical science, Ayurveda, the science of life, and used the same term to

denote veterinary science. This evidences the fundamental unity of the medical sciences.

The evolution of veterinary science is well discussed by Leclainche (12), Smith (13) and by Smithcors (11). Leclainche mentions for example, manuscripts in the library at Upsala pertaining to veterinary medicine published in the 14th century. At this period in certain monasteries human and veterinary medicine was taught and practiced.

The horse, the only means of transportation, and animal plagues threatening man's source of food played an important role in furthering veterinary science. The number of wars in the 15th and 16th centuries emphasized the importance and care of the horse and demonstrated the need of a "horse doctor" attached to the army. At least in the French Army this role was assigned to the riding master who was designated "médecin d'ecuyer" and given the status of a Maréchal.

The veterinary science evolved in a manner comparable to human medicine. It was subjected to sorcery, witchcraft, influence of the Church, the use of weird concoctions, notably dung and urine. In many periods animals, as well as, human illnesses were not attended primarily by members of a professional group. I refer to the period of the riding master, cow leech, the farrier, the quack horse doctor.

At a later period its association with medical science became less—a wide gap existed between the two sciences. In the beginning of the development of veterinary medicine on a scientific basis support from the medical profession was apparently limited to relatively few physicians of broad vision of the calibre of Linné, Willis, Hunter, Rush.* At this period (the latter part of the 18th century) veterinary medicine was disdainfully regarded by the medical profession. It is recorded (12) that Linné disregarded this disdainful attitude in his efforts in founding the Swedish Veterinary School. Smith (13) in writing of the role of physicians in combating cattle plagues in England early in the 18th century writes: . . . "What marred the work of the physicians was their nervous apprehension of loss of dignity in their profession by being engaged in the study of diseases of animals. They nearly all apologized for debasing their profession. . . ." This disdain of the medical profession was still apparent in the latter part of the 19th century. Edgar (14) for example, wrote (1888) of the "strong barrier of prejudice" between veterinary and human medicine.

The first Veterinary Schools were founded in France, in Lyon in 1763 and in Alfort in 1766. Soon thereafter schools were established in the principal cities throughout Europe. The school in Sweden established in 1775, was through the efforts of Linné (1707–1778). He was much interested in Veterinary sciences and took part in measures for control of animal diseases.* At foundation of the Royal Veterinary College of London (1792), John Hunter and other English physicians and surgeons—Astley Cooper, Abernethy, Brodie, Bell—were intimately associated with the College.

Benjamin Rush (15) (1745–1813) believed that veterinary medicine should be included as part of the education of medical doctors. In 1808 he made the following address to his medical students: "The Duty and Advantages of Studying the Diseases of Domesticated Animals." He expressed a desire that the veterinary sciences be taught at the University of Pennsylvania.

James Mease (16) (1771–1846) another Philadelphia physician was also interested in establishing a school of veterinary medicine. He thought such training should begin in the medical school. Mease had a wide knowledge of diseases of animals. He wrote on rabies of animals and was one of the first to describe, what is now known as Texas fever of cattle.

The Veterinary School of the University of Pennsylvania was founded in 1884 by R. Ship-

* It is to be noted that the first Veterinary School in Lyon in 1763 was founded by Bourgelat, a French riding master. The school in Edinburgh was founded (1824) by William Dick, a farrier who was the son of a farrier.

* This phase historically appears to be less known since he is perhaps best known as a botanist. He was also a naturalist and held the chair of medicine at the University of Upsala.

pen Huidekoper, demonstrator of surgery in the Medical School, after spending one year abroad studying the Veterinary Schools in France and Germany. First year students in the Veterinary School took courses in botany and zoology in the biologic department of the University, and second year students attended courses in general pathology and chemistry in the medical school. Jennings, in order to facilitate his training in veterinary medicine in 1850 in Philadelphia, matriculated in the Philadelphia Medical College, and subsequently gave lectures on veterinary medicine to medical students.

Many instances could be cited of doctors of "universal medicine," students of comparative medicine. Space permits mention of only three: John Hunter (1728–1793), Sir Jonathan Hutchinson (1828–1913), and Sir William Osler (1849–1919).

Hunter took note of the whole animal world. The founding of The Royal Veterinary College of London was largely as a result of the sponsorship of John Hunter who was the "life and soul" of the movement.

Hutchinson was an example of the naturalist who takes to medicine and pathology. He was truly the Leonardo of medicine. At different periods he held major appointments in London hospitals as dermatologist, ophthalmologist, and as surgeon.

Hutchinson was much interested in diseases of animals and free reference is made in his writings to comparative pathology. He was especially interested in heredity as influencing physical characteristics of certain animals. In some of his writings he discussed the influence of the color of certain animals in their proclivity to diseases. Hirschberg (17), in his visit to Hutchinson, writes as follows: "Upon our returning to the manor we were informed that one of the goats had just died. Hutchinson immediately performed an autopsy and demonstrated how vaginal prolapse, although not dangerous in women, may cause death in animals through gangrene caused by constant friction. Hutchinson then discoursed on the difference between human and animal science of healing."

Osler had a wide range of interest—botany, zoology, natural sciences, comparative medicine. As a medical student in Toronto in 1870 he pointed out the presence of trichina spiralis in the muscles of one of the bodies he was dissecting. This was one of the earliest observed cases in Canada. Later as a distinguished pathologist he demonstrated a number of pathologic specimens from animals. He had great interest in study of diseases of animals, to satisfy which he was Professor of Physiology and Pathology in the Veterinary College in Montreal, and Vice-President of the Montreal Veterinary Medical Association.

In conjunction with a veterinary student he published in 1883 a report on the three more common parasites transmissible to man: Trichinella, cysticercus and echinococcus, based on examination of 1037 hogs in an abattoir. When he came to Philadelphia about the year the Veterinary School of the University of Pennsylvania was established, he continued to demonstrate pathologic specimens from animals at meetings of the Pathologic Society of Philadelphia and published papers in the American Journal of Comparative Medicine and Surgery.

REUNION OF HUMAN AND VETERINARY MEDICINE

Toward the end of the 19th century, the gap between the two sciences began to become less. This became more apparent in the beginning of the 20th century, prejudice disappeared, the two sciences become more closely united. In addition to the aforementioned circumstances in the evolution of the interrelation of human and veterinary medicine the influence of the following circumstances may be cited.

There are numerous examples in the history of medical researches of the reciprocal influences of human and veterinary physiology, pathology and infectious diseases that had great import in the advance of knowledge of human disease.

Jenner's experiment afforded the first proof of immunization against an infection. The first definite proof of bacterial origin of a disease was the result of Koch's study of anthrax (1876)—a disease of animals. The discovery in 1897 by Loeffler and Frosch of a viral cause of foot and mouth disease was one of the first of the filtrable viruses to be incriminated as a cause of disease in man or animals. There is an intimate relationship in our knowledge of human bovine tuberculosis. The demonstration that Texas fever of cattle by Smith and Kilborne (18) (1893) was caused by a protozoan parasite paved the way to an understanding of the cause and transmission of such diseases as yellow fever, Rocky Mountain spotted fever, typhus and malaria. An effective treatment of hookworm in man was founded on Hall's (1921) study of a variety of anthelmintics in treatment of hookworm in dogs. Knowledge of the human form of Erysipelothrix rhusiopathiae infection, the septicemic form of which may end fatally (19), is entirely founded on studies in the domain of veterinary medicine of the infection in swine. (The causal agent was isolated by Loeffler in 1882.) Eosinophile granuloma of cats—so-called syphiloid and mast cell tumors of dogs has aided in visualization of the histocytoses in man. Laidlow and Dunkin's (20) study (1928) of distemper has been introductory to a wider field of research in viruses and may have possible analogies with human influenza.

Knowledge of comparative medicine is helpful to research in human medicine. I refer to investigative work of diseases of man that occur in animals. The cat, for example, as an experimental subject for study of the histocytoses. The dog for study of the pathogenesis of eczema and pruritus. The use of certain animals for study of phytophotodermatitis. Certain dogs and grey horses for study of malignant melanoma. Why do animals eat their placenta? In the domain of immunology—why is the swine so susceptible to naturally occurring infection with Erysipelothrix rhusiopathiae but highly resistant to experimental inoculation? Why are the white mouse and pigeon so susceptible and the field mouse entirely immune?

The vital importance of the zoonoses—diseases which are transmitted naturally between vertebrate animals and man (21) emphasize the close relation of human and veterinary medicine. These diseases not only unfavorably affect the health of man, but through the serious economic effects of some of these diseases, man's food supply is also affected. Brucellosis and foot and mouth disease are notable examples.

It is generally stated that there are 86 different diseases comprising the zoonoses. There is evidence that this list is increasing. Not all of the zoonoses are major health problems, the twelve major ones are recommended by the World Health Organization to be made notifiable throughout the world.

These diseases so important to veterinarians are of equal importance to medical doctors. Knowledge of these diseases—the "prepared mind" and a good epidemiologic history leads to diagnosis—to wit, a patient with pneumonitis among diagnostic possibilities, psittacosis should be included. Similar implications apply to onset of symptoms following contact with a variety of animals, poultry and pigeons, the bite of a cat, a bat, skinning a squirrel, a rabbit, contact with dried egg powder, fresh duck eggs, or from a cut finger of an abattoir worker.*

Prior to Second World War the role of the veterinarian was essentially confined to milk and food sanitation. Since this era the important role the veterinarian has always played in public health has been more fully appreciated. Until recently 23 States now have a veterinarian full time staff member of departments of public health.

This recognized position in public health is amply rationalized in consideration of the vital

* Fiessinger and Brouet's patient (Presse méd **42:** 889, 1934) with Erysipelothrix rhusiopathiae septicemia, had hematoma like swelling of the ears. Diagnosis was facilitated by a veterinarian student who called attention to similar occurrence in swine with the septicemic form of infection.

importance of the zoonoses and the potential contribution of veterinarian medicine to public health. The role of the veterinarian in combating the animal source of zoonotic disease becomes apparent. This task is essentially in the domain of veterinary medicine—only a few illustrations need be given. It is estimated that for every rabid dog in a given vicinity 20 persons receive the Pasteur treatment. It is also estimated that there are 10,000 human cases of brucellosis yearly and that one-third of abattoir workers have significant brucellosis blood titers. Twenty per cent of Americans have Trichinella larva in their muscles.

The change in teaching as discussed by Mueller (9) and by Dale (10) is becoming apparent. Some schools of veterinary medicine in Great Britain—Edinburgh, Cambridge and Glasgow are under the faculty of medicine. Medicine, dental and veterinary students attend the same classes in the first year of instruction. In these institutions there is close interdepartmental relation of the medical and veterinary school.*

There is other evidence of increasing correlation in teaching of human and veterinary medicine. At the Veterinary School of Medicine of the University of Pennsylvania such correlation exists in pathology, biochemistry, and until recently, in virology. A faculty member of the Veterinary School teaches comparative dermatology in the Graduate School of Medicine. Such correlation exists in two of the thirteen veterinary schools in the United States.†

Perhaps another milestone in the liaison of teaching human and veterinary medicine will be in graduate teaching. This is becoming apparent. In 1940 Harvard University initiated the eligibility of graduates in veterinary medicine for admission to the Harvard School of Public Health on the same basis as graduates in medicine. Through the affiliation of the Veterinary School of Medicine of the University of Pennsylvania with the Graduate School of Medicine graduate work is now available to veterinarians.

I believe one can envisage teaching of comparative aspects of specialized subjects in graduate courses; such as, pathology, parasitology, dermatology, ophthalmology, and courses in certain subjects for veterinarians; such as, pathology, microbiology, public health, radiology.

As further evidence of the close relation of the two professions may be cited the collaboration of the veterinarian in experimental surgery and in human surgery in relation to transfer of healthy blood vessels and organs from one person to another. The use of the Stader splint originally intended for animals is now used for reduction of fractures in human beings. The development of dicoumerol originated from the observation of the veterinarian of the action of sweet clover poisoning in animals. Dr. Fred O'Flaherty, a veterinarian of the Research Laboratory of The Tanner's Council of America, University of Cincinnati, has contributed considerably to knowledge of chemistry of hides and skin. The functioning of veterinarians as pathologists and microbiologists in medical hospitals may be cited as additional evidence.

One can envisage a still closer association of the two professions than now exists. The sharing of many courses of students in veterinary medicine with medical students. A greater expansion of comparative medicine in graduate teaching. The creation of chairs of comparative medicine and sections in national medical organizations. A greater number of symposia and conferences pertaining to comparative medicine and admissions of veterinarians in medical societies and organizations. In this manner the veterinarian will have more intimate contact with his medical confreres to the advantage of both. I am in accord with Mueller (9) who writes, "The veterinarian has far greater knowledge of human sicknesses than the average medical doctor does of animal

* From information obtained in visit to these schools.

† From information obtained in questionnaire sent to these schools.

sicknesses. . . . One may not blame medical doctors for this lamentable ignorance on their part. It is simply the result of that obsolete concept of division: Man and animal."

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Frederick G. Novy and Some Origins of American Bacteriology[1]

By ESMOND R. LONG, PH.D., SC.D., M.D.[2]

WHEN a distinguished teacher and investigator of bacteriology reaches the age of almost 93 before leaving the scene of his labors, his passing merits more than ordinary attention, for his nine decades have encompassed all the years of evolution of his science. A 90 year old bacteriologist in 1957 has seen virtually the entire sequence of events that have led to our present understanding of the cause of infectious disease and the role of bacteria and viruses in human illness, animal husbandry, agriculture and industry.

On August 8 of this year occurred the death at 92 years and 8 months of Frederick G. Novy, one of the last of the American pioneers in microbiology who studied in the laboratories of Pasteur and Koch and worked personally with the leading pupils of these founders of bacteriology. In the summer of 1888 Novy, an instructor in hygiene in the University of Michigan, and Victor C. Vaughan, his chief mentor in its medical school, influenced by the rising tide of reports on the agents of communicable disease, and aware of the need to introduce courses in the subject for students in the medical school, journeyed to Europe to learn new facts and new methods of study in the rapidly developing field. In Berlin, their destination, Novy worked primarily under Koch's assistant Carl Fraenkel, but listened to lectures by Koch himself. Back in Ann Arbor in the fall he made preparations for a course in bacteriology, which was officially offered as a three month elective in the University in January 1889. Relatively few students took the course, but some of the faculty attended. Among these was the professor of physiology, Henry Sewall, who knew he had tuberculosis and wished to study his own case by the new bacteriological technics. Sewall was one of the first to appreciate Novy's talents and foresight. So successful was the course that in the fall of the next year, 1890, bacteriology became a requirement for students in the medical curriculum.

Novy made many subsequent trips to Europe to improve his understanding and acquaintance. He studied at the famous University of Prague, always a leading center for pathology, in 1894, and at the Pasteur Institute in Paris in 1897, two years after Pasteur's death. Although he missed an association with the French master, Novy became an intimate of the Pasteur Institute and developed a warm and lasting friendship with an early successor of Pasteur, Emile Roux, one of the discoverers of diphtheria toxin.

Novy grew rapidly in stature as teacher and investigator. By 1900 his place as a foremost bacteriologist in this country was well established. He had published a textbook for students, collaborated with Vaughan in a widely read volume on ptomaines, taken part in local studies on typhoid fever, written authoritatively on the hog cholera bacillus, discovered one of the several etiological agents of malignant edema, developed apparatus and methods for investigating the biology of anaerobic bacteria, and written extensively on laboratory methods in general bacteriology. In 1900, when suspicion was raised by a report from an officer of the U. S. Marine Hospital Service that bubonic plague was smoldering in San Francisco's Chinatown, Novy was a logical choice for membership in a commission ap-

[1] Read before the Philadelphia Clinical Meeting of the American Medical Association, 4 December 1957.

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pointed by the Secretary of the Treasury—whose responsibilities then included what is now the Public Health Service—to ascertain if the disease really was rife. The Commission, consisting of Simon Flexner, Chairman, Llewellys F. Barker and Novy, located several cases of bubonic plague, proving conclusively that the disease was endemic, and opened the way for rigid measures for the prevention of its spread.

In the same year, 1900, Novy took part in the foundation of the Society of American Bacteriologists; in 1904 he became its fifth president, succeeding Theobald Smith in that influential position. Two years previously, in 1902, he had been made full professor and head of the now well established Department of Bacteriology in the University of Michigan.

NOVY'S EDUCATION AND EARLY YEARS

Novy was born in Chicago December 9, 1864, of immigrant Bohemian parentage. He attended Chicago public schools, and rapidly developed an interest in science. In this he was stimulated greatly by his high school chemistry teacher, Marc de la Fontaine, an independent investigator, as well as teacher. Like so many boys who later became eminent in biological fields, the young Novy was fascinated by the opportunities of his environment, and constantly sought ways of improving his understanding of it. De la Fontaine had shown him how to use a microscope, and nothing would satisfy Novy until he had an instrument of his own. He worked nights in the Chicago Public Library to secure the necessary money, and after he acquired the coveted tool he began, with intense enthusiasm, to search ponds and fields near and far for life invisible to the naked eye. His interest in microscopy was permanent; in later years it led directly into the bacteriology he did so much to develop. At the time, however, it was an avocation; his real purpose was to follow in the footsteps of his teacher, de la Fontaine, and be a chemist. De la Fontaine was an admirer of Albert B. Prescott, Professor of Organic Chemistry at the University of Michigan, and to the latter Novy wrote for information on courses in chemistry at that University. So impressed was Novy by Prescott's replies that he became eager to enter the University of Michigan. His extraordinarily understanding and receptive parents met this wish by themselves moving to Ann Arbor and starting life again in that city. There young Novy could pursue his college course while living at home. It remained his own home for seventy-five years.

Novy graduated from Michigan, after specializing in chemistry, in 1886. He looked briefly for a job elsewhere, but immediately closed with an offer from his teacher Dr. Prescott of a modest post in the University's Department of Organic Chemistry. He began work immediately for a Master's degree and attained this in June 1887, with a thesis on cocaine and its derivatives. In that year Victor Vaughan persuaded him to take a more responsible position in the University as Instructor in Hygiene. Thus began a long association, marked by close collaboration and numerous joint publications, of which the best known was their book on *Cellular Toxins*, which had started out as a smaller work on *Ptomaines and Leucomaines, or the Putrefactive and Physiological Alkaloids*. As we have seen, Vaughan and Novy journeyed to Europe together to study bacteriology; it was Novy who first brought up the idea of the trip. A student always, Novy followed the medical curriculum himself, as rapidly as he could in the midst of his academic responsibilities, and received the M.D. degree in 1891. In that year he was made Assistant Professor of Hygiene under Vaughan. The latter became Dean of the Medical School that year, so that much of the professional responsibility for the Department devolved on Novy. This early obligation, imposed when he was less than 30 years of age, threw him into the midst of a highly stimulating group, vividly concerned in the development of medical instruction in the Middle West.

EARLY AMERICAN BACTERIOLOGY

At that time there was little original bacteriology in the United States. A few investigators, however, had made fundamental discoveries. Thomas J. Burrill (1839–1916), professor of botany and horticulture at the University of Illinois, had discovered the bacterial cause of pear blight in 1877, and had introduced a few lectures on basic bacteriology in his course in botany at that University.

Studies of far-reaching significance for the entire field of microbiology were the investigations of Theobald Smith (1859–1934) and his associate F. L. Kilbourne at the Bureau of Animal Industry of the U. S. Department of Agriculture on the etiology and transmission of Texas fever, in which the parasite *Pyrosoma bigeminum* and its transmitting agent, *Boöphilus bovis*, were discovered. Theobald Smith continued as one of America's foremost bacteriologists to the end of his life. Among his notable early contributions was the differentiation, in 1898, of the bacilli of human and bovine tuberculosis.

Theobald Smith's early work was carried out at the Bureau of Animal Industry in Washington. The Bureau, under Daniel E. Salmon, was already (1879) deeply engaged in fundamental problems in the bacteriology of animal disease. Salmon was an effective teacher, whose lasting influence on bacteriological science was later recognized in the official designation of the *Salmonella* group of organisms.

Of all of the early American group the one who came the closest to the fast moving German and French schools in methods of study and types of achievement, was George M. Sternberg (1838–1915), U. S. Army Medical Corps officer, and in his later years Surgeon General of the Army. Sternberg is best known as one of the several discoverers of the pneumococcus (1881), an organism of wide biological diversity which was identified by several of the early workers under different names. Sternberg was an investigator in many fields of microbiology, a systematist and authoritative teacher. For years his *Manual of Bacteriology*, originally a translation and extension of a volume by the French bacteriologist Antoine Magnin (1880), was the standard text in the subject in this country.

In the late 1870's William H. Welch and T. Mitchell Prudden were studying and demonstrating methods of bacteriology in New York, Welch at the Bellevue Hospital Medical College and Prudden at the College of Physicians and Surgeons. Later, at Johns Hopkins University, Welch organized the first truly modern school of medicine in this country, a monumental advance in American science. He achieved lasting fame in the history of bacteriology for his discovery, in cooperation with George H. F. Nuttall, of the gas bacillus, later known as *B. welchii*, in 1892.

In the late eighties and early nineties courses and investigative laboratories for bacteriology were springing up throughout the country: under Francis Delafield, T. Mitchell Prudden, Hermann M. Biggs and William Hallock Park in New York, under men like Bayard Holmes, William T. Belfield and Henry Gradle in Chicago, under the biologists William Trelease, Edward A. Birge, and Harry Luman Russell at the University of Wisconsin, and under such men as Alexander C. Abbott, Henry Formad, Ernest La Place, Lawrence F. Flick, Samuel G. Dixon and Joseph McFarland in Philadelphia, Frederick P. Gorham at Brown University, and L. H. Pammel at the Iowa State College of Agriculture at Ames.

Many of these men had studied in Europe and brought German and French training and methods to the United States. Welch had studied under Julius Cohnheim at Breslau, and under his tutelage had become an independent investigator. Others were avid readers of the German literature, quick to repeat the work of which they had read. Still others were rapidly trained, and made into specialists, by the early pioneers who had returned from Germany; e.g., Welch trained H. L. Russell and Hermann Biggs stimulated William Hallock Park.

Behind the Europeans in the development of

basic facts and fundamental principles in bacteriological science, Americans were not slow in practical application of the scientific facts established. In public health Charles V. Chapin at Providence, Rhode Island, William T. Sedgwick in Boston, and Hermann M. Biggs and William H. Park in New York—to mention but a few—had put this country in the front rank in improvement of sanitation and control of infectious disease through an understanding of bacteriology. The Hygienic Laboratory of the U. S. Public Health Service, now the National Institutes of Health, began its distinguished career in 1901.

In medicine the principles of bacteriology were taught by many clinicians in the late 80's, and in surgery by as rough and ready a practitioner as Nicholas Senn. John Buchanan brought out "An Encyclopaedia on the Practice of Medicine Based on Bacteriology" as early as 1890.

Bacteriology was tied closely to pathological teaching in this country by Francis Delafield and T. Mitchell Prudden, Simon Flexner and L. Emmet Holt, Welch and Nuttall, William T. Councilman, Harold C. Ernst and Frank B. Mallory. The indissoluble relations of the two sciences were recognized in the foundation of the American Association of Pathologists and Bacteriologists, of which Councilman was the first president, in 1901. Many fundamental relations of bacteriology with immunology were developed by Ludvig Hektoen in Chicago in the closing years of the nineteenth century.

Veranus A. Moore (1859–1931) at Cornell University pioneered in veterinary bacteriology. His "Laboratory Directions for Beginners in Bacteriology" (1898) was a forerunner of many publications in this field. In agricultural and dairy bacteriology Herbert W. Conn of Middletown, Connecticut, was an early and continuing leader, and, as we have seen, a strong school developed in this field at the University of Wisconsin. The influence of the Bureau of Animal Industry of the U. S. Department of Agriculture was profound in all these fields.

The first society formally and specifically devoted to Bacteriology was the Society of American Bacteriologists, established in 1899; its founders included many of the men already mentioned, including Frederick G. Novy. Alexander C. Abbott, Edwin O. Jordan and Herbert W. Conn were prime instigators in its organization.

At the turn of the century numerous American journals were devoting all or much of their space to bacteriological developments; these included the *Journal of Medical Research* (founded as the *Boston Journal of the Medical Sciences* in 1896), the *Journal of Experimental Medicine*, also founded in 1896, and the *Journal of Infectious Diseases*, founded in 1904. The *American Journal of Public Health*, which went through a series of transitions in name and format before its present stabilization, was from its earliest years a medium for publication of the applications of bacteriology to public health.

NOVY'S CONTRIBUTIONS TO MICROBIOLOGY

Frederick Novy was an early entrant in the environment briefly sketched in the foregoing paragraphs. In the early 90's he put out a short informal text on *Laboratory Work in Bacteriology*, which was elaborated, revised and enlarged in a second edition in 1899. It came out through Novy's insistence, as noted in the preface of the book, that a thorough course of laboratory instruction in bacteriology was essential in the proper education of medical students. The text was based on the day to day work of the Hygienic Laboratory of the University of Michigan and represented the thorough teaching characterizing all of Novy's efforts.

Although Novy began his professional life as a chemist, and published a chemical text for medical students (*Laboratory Work in Physiological Chemistry*, 1892) his early responsibilities in hygiene and public health, fostered by Victor Vaughan, and his own self-acquired training in microscopic zoology, soon led him into a career in microbiology. His association with Vaughan and his own interest in chemistry rapidly carried him into a search

for toxic products of bacterial growth. As early as 1890 he reported on poisonous products he had discovered in cultures of the hog cholera bacillus. In 1892 he was busy in the study of attenuation of virulent microorganisms. In 1893 he announced the discovery of "a new anaerobic bacillus of malignant oedema," a variant of the well known organism discovered by August Gärtner. Throughout the 90's he was vigorously engaged in acquainting the Michigan public with facts on the germ theory of disease, on the nature of diphtheria, typhoid fever and tuberculosis, on food poisoning, on disinfection, and on many similar matters of significance for public health.

Shortly after the turn of the century he began the work for which he is best known, the study of trypanosomes and spirochetes. His first ambition was to cultivate trypanosomes in artificial culture media. In this, in association with an assistant, Ward J. MacNeal, he was soon successful. They used the rat trypanosomes *T. lewisi* and *T. brucei*. Their results met prompt interest and early confirmation abroad. The distinguished Charles Nicolle, Director of the Pasteur Institute of Tunis, introduced a modification of their blood agar medium, which became known as the N:N:N medium (after Novy, MacNeal and Nicolle, on the mistaken assumption abroad that MacNeal's last name was Neal). Novy's work on trypanosomes quite naturally carried him on to investigations on the spirilla and spirochetes. So intense was his devotion to this field, and so well known in the medical school were his studies, that he acquired the nickname 'Spi', and Spi Novy he remained to friends and students the rest of his life. He had a famous controversy with Fritz Schaudinn, before the latter's discovery of the spirochete of syphilis, and corrected a biological misconception to which the German protozoologist was dedicated. Novy himself discovered the causative agent of American relapsing fever, to which the designation *Spirocheta novyi* was later quite properly attached. He made extensive studies on related organisms, including the spirochete discovered by Obermeier, and studied and cultivated several other species of spirilla and trypanosomes. All of this work earned a position of leadership in this branch of microbiology. He pioneered also in the study of viruses through investigations of the filtrable agent of rat virus disease.

He was perennially interested in problems of resistance to bacterial infection and the general field of immunology. His concern in the latter domain led him into a venturesome, intense attack on the problem of anaphylaxis. The mechanism of the phenomenon, which had been brought to the attention of the medical world largely by the work of the French physiologist Charles Richet, was, as it still is, a puzzle. The experiments of Novy and his assistant Paul de Kruif in 1917, led them to postulate the existence of a specific poison, such as other investigators had envisioned under the designation anaphylatoxin. Their experiments were the outgrowth of attempts to immunize animals against trypanosomes. The violent reactions that took place on repeated injections were obviously of the anaphylactic type and led Novy and De Kruif to believe the trypanosome procedure offered a new tool for more intimate understanding. The speed of the reaction, the minuteness of the amount of trypanosomal material capable of eliciting it, and the discovery that toxic properties could be induced in rat serum by a few minutes' incubation with the antigen, when taken together, convinced the investigators that they were dealing with a powerful poison. The duplication of results by mere incubation of serum with other colloids indicated the complexity of the problem. The nature of the poison was not determined, but the results ultimately became part of the large body of evidence implicating histidine in anaphylactic shock.

Novy's last outstanding contribution to bacteriological science was his introduction of procedures for studying the respiratory metabolism of bacteria. With Malcolm H. Soule he published a series of investigations on gas exchange in the respiration of tubercle bacilli (1925) that long remained a model in this field.

Such studies today are carried out by the more delicate machinery of the Warburg apparatus or similar equipment, but no greater precision is attained than that achieved by Novy and Soule with Novy's more cumbersome but effective devices.

In his later years Novy suffered the fate that seems inevitable for men who have proven their organizing ability by success in research, viz., gradual transition from scientific investigations to the confining tasks of administration. In 1935, when he retired from all his official academic duties, he was not only head of the Department of Bacteriology but Dean of the Medical School, trusted and respected by all, and beloved by students and faculty alike.

Novy considered himself an old time bacteriologist and always listed himself as such. The fields of his greatest achievement were not strictly in bacteriology, however, but rather in the broader domain of microbiology. His work encompassed several of its recognized major branches, true bacteriology, protozoology, virology and immunology. In his fifty years of intense professional activity he had seen remarkable developments in understanding in all of these fields. In the twenty-two years of retirement, he remained a recognized authority and wise counselor, whose advice and assistance were constantly in demand.

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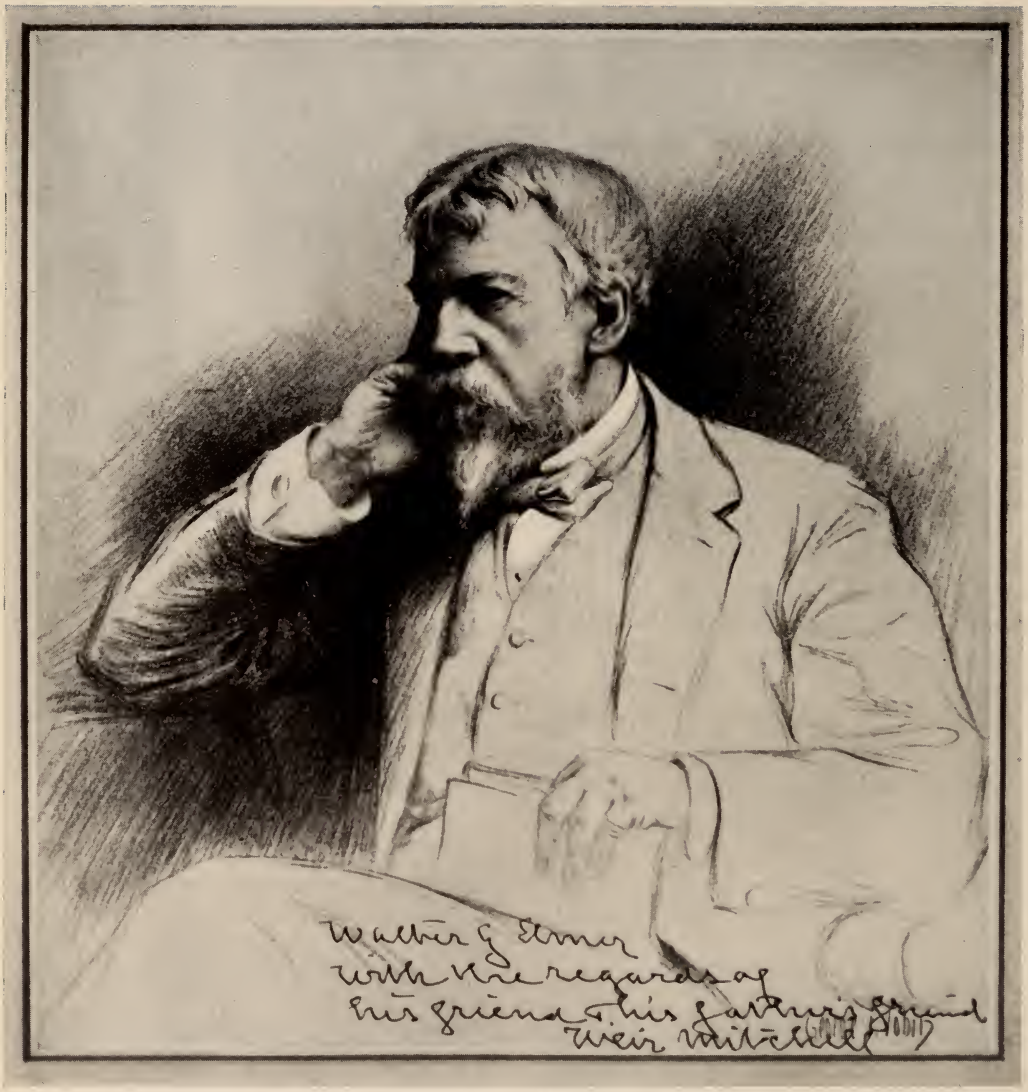

Fig. 1. Original pencil drawing of Dr. S. Weir Mitchell by George T. Tobin (1864–1956)

Some Personal Recollections of Dr. S. Weir Mitchell[1]

By WALTER G. ELMER, M.D.[2]

THOUGHT TRANSFERENCE AND RELATED SUBJECTS

WE were sitting in comfortable arm chairs before the open wood fire in Dr. Mitchell's office in his home, 1524 Walnut Street in Philadelphia. It was midwinter, the streets were cold and windy and we enjoyed the warmth and shelter of the room. We had been discussing thought transference. I asked Dr. Mitchell if in his own experience he had ever been present at a genuine, undoubted demonstration of telepathy. His face lighted up and there was just the trace of a smile as he recollected his tilt with Charcot. He leaned forward and gazed into the fire in silence, his features glowing clearly in the fire-light. "Well, it was like this", he said. "Mrs. Mitchell and I were staying in Paris. Professor Charcot and I had been close personal friends for many years. When he learned that we were in Paris he invited me to attend one of his lectures when he proposed to demonstrate thought transference to his audience. I gladly accepted his invitation.

"The clinical amphitheatre was filled with students and visiting doctors. Charcot introduced me to the audience, a chair was brought in for me and placed on the floor of the amphitheatre. Charcot then proceeded with his lecture and said he proposed to demonstrate that mental commands can be conveyed to a subject in the hypnotic state. A nurse brought in a young girl who had been hypnotized. She was in a wheel-chair and was turned around with her back to the audience.

"Professor Charcot stood facing the audience with his back to his subject as her back was to him.

"In order that his audience might be able to understand what his mental commands were, he would make certain movements with his arms—both arms horizontal, both arms vertical, one arm horizontal and one vertical, then just the reverse and others to follow for quite a succession of gestures. If these mental commands were received by his subject, she would exactly duplicate the gestures. And this the girl did precisely—every one of the professor's movements. When Charcot brought his lecture to a close he seemed much pleased with the success of his demonstration. And his audience—as the doctors and students filed out of the amphitheatre—seemed duly impressed. Then Charcot turned to me and, smiling, said—'Well, Mitchell, what do you think of my experiment?' I said 'Why, Charcot, that girl could see.'

'See! What do you mean? '

'I sat beside her, watching her very closely. She gazed straight ahead with half closed eyelids at the swinging glass doors that lead into the clinic. These glass doors are backed by green baize curtains and make perfect mirrors. The girl saw your reflection and exactly followed each of your movements.'

"Charcot flushed and was angry. He said—'Mitchell, I am always glad to have you as my guest, but I do not wish you to criticise my methods.' He apparently forgave me later."

Here were two great men—face to face. One the leading authority on nervous diseases in Europe, the other the leading authority on nervous diseases in America.

Dr. Mitchell got up to put a log on the fire.

"Then there is the case of Mrs. Piper," I said.

"Yes, there is the case of Mrs. Piper. I

[1] Read by title, Section on Medical History, College of Physicians of Philadelphia, 11 March 1958.

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followed her career with a great deal of interest. It was reported that she had carried out quite a number of successful experiments with the London Society for Psychical Research and elsewhere throughout England and Scotland. Her supposed communications with the spirit world everywhere created a profound impression.

"When she returned to this country, I wrote to her asking her if she were willing to come to Philadelphia for an interview. She replied that she would be glad to come. I then asked my son John to be present.

"We received Mrs. Piper in the front hall as a distinguished visitor and ushered her into my office. I locked the door. After a pleasant conversation, I asked her if she would be willing to communicate with the spirit of my brother. She replied that she was quite willing to try. I told her my brother had died a tragic death. Could she tell me his name and also when and where and how he died. The three of us then bowed our heads and sat in profound silence, nothing but the ticking of the hall clock and the distant rumble of the city. The minutes dragged on—

"Finally, Mrs. Piper began to speak: 'John'; a pause; 'Henry'; a pause; 'Thomas'; 'Charles, Andrew, James,' and on through quite a number of names.

"Then after a pause—'I see a ship. The ship is in a storm at sea. The storm grows worse and the ship is in great distress—Yes, the ship is sinking, it has gone down. Your brother was lost at sea.' She stopped, and after a little while looked up.

" 'My brother's name was Robert,' I said, 'and he was killed in the battle of Antietam.' We tried again and still again. Each time she failed. She apologized for her failure and explained that she was confronted by such powerful antagonism and skepticism that she was utterly unable to carry on. We thanked her for her willingness to come and make the effort and we parted good friends."

One evening I called at the house and inquired of the footman if Dr. and Mrs. Mitchell were at home. Dr. Mitchell heard my voice in the hall and called out "Oh Elmer, come in. Show Dr. Elmer in." And I was ushered into Dr. Mitchell's office. He rose to greet me—"I was sitting here reading; Mrs. Mitchell has gone upstairs. I am glad you came. Take this chair and have one of these cigars—my special brand." Dr. Mitchell was so genial and friendly, it was always a delightful experience to be with him.

"I would like to hear more about the Seybert Commission," I said.

"We had interesting meetings and almost always something worth-while came out of them. I recall the slate-writing episode. The medium proposed to communicate with a departed spirit who would write a message on a slate to one of us. A double slate was produced—the kind made of two parts which fold together such as school children use. A slate pencil was placed in the slate, which was closed and tied firmly with a heavy cord. The medium and three of us sat at a table. I was sitting opposite the medium. He held the slate on his knees under the table. The lights were turned low. We sat with an elbow on the table, forehead resting on hand. I cautiously withdrew a small mirror from my pocket which I held under the table. We sat in silence, looking down, in deep thought. After several minutes we heard the pencil scratching on the slate. As I looked down in the mirror, held at an angle, I could see the medium's hands quickly untieing the cord, opening the slate, writing with the slate pencil, closing the slate and retieing it with the cord. After a pause, he looked up, brought up the slate and placed it on the table. The cord was untied, the slate opened and there was the message from the spirit world.

"We were all duly impressed."

Dr. Mitchell continued—"Table turning is something entirely different and it is very difficult to convince those who are taking part in the experiment of what is actually happening. Several persons sit around a table with their hands resting lightly upon the table, waiting silently for a spirit or spirits to cause the table to move. After several minutes of suspense the table does begin to move and

those taking part are convinced that the table is drawing their hands along with it. They are unwilling to believe that the reverse is true.

"I had provided myself with a number of hat-pins. After the subjects had placed their hands upon the table I stuck a hat-pin into the table between the fingers of each hand of each person. The pins were all vertical. But after a while they all began very gradually slanting to the right and the table began turning to the right, indicating that the hands were pushing the table. The table was not dragging the hands along with it. But no one had the slightest idea that he was taking an active part in moving the table."

"Dr. Mitchell, 'Constance Trescott' is a most interesting and deeply absorbing novel."

"I am glad you think so. I was on my way to New York one day and was seated in the railway car beside a young lady who was intently reading a book. I glanced down and saw that it was 'Constance Trescott.' I asked her how she liked the book she was reading. She looked up and said 'It is a strange story and I am not sure that I like it and I have been wondering why Dr. Mitchell wrote it.' However, I did not tell her that I was the author."

"In the novel you describe one of your characters as having received a severe mental shock which was followed by the illusion of seeing a smiling face as through a veil or mist—a little above and to the right. Whenever he looked up it was there. When he looked down it was gone. It persisted for a number of days and then gradually faded. Have you ever had a patient who revealed that peculiar symptom?"

"No, not a patient, but it was my own experience. Phillips Brooks and I were very close personal friends for years. His loyal friendship and congenial companionship meant a great deal to me. We spent many hours together. I was descending the stairs in my home one morning when someone called to me, 'Weir, have you heard that Phillips Brooks is dead?' I was severely shocked—I had not learned of any illness—I stopped on the landing of the stairs holding onto the balustrade for support, then slowly came down to the first floor and stood there looking down at the floor and trying to realize what had happened. Then, as I looked up, I saw the smiling countenance of Phillips Brooks. It appeared to be rather silvery, shimmering, seen through a mist or veil. When I looked down it vanished. When I looked up it was there—a little above and to the right. It followed me everywhere for about ten days—then gradually faded and was lost."

We were having tea in the library. Mrs. Mitchell was telling me about their trip to Florida, from which they had just returned.

"When Dr. Mitchell comes in—he always comes in for tea every afternoon about five o'clock—you must ask him to tell you about having his mind read while we were staying at the Jekyl Island Club on our way home."

Dr. Mitchell came into the room to join us at tea. He sat down and taking a cup of tea from Mrs. Mitchell—"It is a relief after being busy all day to come in for a little relaxation and a refreshing cup of tea. It braces me up and I always feel the better for it."

"Weir, I have been telling Dr. Elmer about our trip to Florida. Now you must tell him how you had your mind read while we were staying at the Jekyl Island Club."

Dr. Mitchell smiled in a good-humored way—"Well, my dear, the young lady was supposed to have read my mind and I must confess that I am still unable to understand how she apparently accomplished it.

"Three or four members of the club and myself—we were all intimate friends—were sitting on the club piazza one evening after dinner. It had already grown dark. One of them said, 'Weir, have you had your mind read yet?'

" 'My mind read? Why no, of course not. Who is going to read my mind?' "

" 'Well, we have all had our minds read. Why don't you? Miss Mary A., from New York, is staying here with some of her family and she has been reading our minds.' "

" 'That's all nonsense. I don't believe it.' "

" 'Are you willing to let her try?' "

" 'Certainly. She may try all she likes.' "

" 'I'll go in and see if I can find her.' "

"He appeared shortly, leading Miss A. by the hand.

" 'Well, my child, so you have been brought out here to read my mind, just as you have read the minds of our friends here. Now go right ahead and try it on me.' "

" 'Oh, but Dr. Mitchell, I cannot read *your* mind. I am really too frightened to try.' "

" 'What is there to be afraid of? I am just like everybody else. I will do exactly what you tell me to do.' "

" 'I must get a piece of paper and pencil.' "

"She went into the club and reappeared with pencil and paper which she handed to me."

" 'I will go into the club while you write something on the paper, fold it up and then call me back.' "

"I wrote, 'Wild Eagle of the Western Skies, Why are your feathers red?' It was the first line of one of my poems, supposed to be an ominous foreboding of impending war. I folded the paper and called her back and she came out and joined us."

" 'Oh, but you must fold it into a very small packet. Look, I'll show you.' "

"She took the paper and folded it into the small packet she had described—not more than a half-inch square."

" 'Now you must press it against your forehead directly between your eyebrows like this and then look down and sit very still and concentrate on what you have written.' "

"She handed me the paper which I held against my forehead as she had directed and looked down and concentrated my thoughts on the writing. She sat in front of me looking down."

" 'Now think of the first letter of the first word . . . the second letter . . . the third letter . . . the fourth letter. Now think of the first letter of the second word . . . the second letter . . . the third letter . . . the fourth letter . . . the fifth letter. Now the first letter of the third word. . . .' "

"It occurred to me at this point that she was correctly spacing the letters in the words. And so it continued on to the end of the sentence. Then with an expression which might have indicated the great mental effort she had been making, she looked up and spoke slowly—'You have written—Wild Eagle of the Western Skies, Why are your—I am not quite sure of this word. I am rather confused but the last word is red.' "

" 'Wild Eagle of the Western Skies, Why are your feathers red? Yes, child, you have it—all but the one word.' "

" 'Oh, did I get it right? Let me see.' "

"She took the paper and unfolded it and there was the sentence. Now I am still at a loss to understand how she accomplished it. There was a swinging lantern hanging from the roof of the veranda but the light was so dim she could not have read the sentence even if it had been held directly before her."

"Dr. Mitchell, do you believe that thought transference is possible?"

"Yes, I do. I believe one person's mind can influence another person's mind in ways which are outside of all known physical laws and in ways which we do not understand."

Mrs. Mitchell sat quietly sipping her tea—an interested listener.

A few days later, I was invited to come out to the T.'s country house for dinner and to stay over night. An attractive house, surrounded by large grounds and fine old oak trees. At dinner were Mr. and Mrs. T., Miss T., and Miss Mary A., who was staying in the house.

During the course of the conversation, Mr. T. turned to me and asked—"Dr. Elmer, have you ever had your mind read?"

"No, I haven't. Why do you ask?"

"Because Mary will read your mind if you will let her. She is really remarkable. She has read Mrs. T.'s mind, our daughter's, and mine since she came here two days ago. She has just had a letter from her father in New York enclosing a clipping from the *New York Herald* and stating that no daughter of his could read *his* mind, and he forbade her to go on with these experiments."

The article from the *New York Herald* was a

description of the annual meeting of the Association of American Physicians held in Washington and of Dr. Mitchell's address on the fear of cats. At the end of his address, Dr. Mitchell commented—"I have just had the unusual experience of what was presumed to be reading of my mind. I confess I am still at a loss to understand how this young lady, whose family is well known to me, carried out this experiment. I will give the matter very careful consideration and report to you at some later date."

This aroused my curiosity. Why should so distinguished a person as Dr. Mitchell make this comment before so distinguished an audience?

As we were leaving the dining room after dinner, Miss A. and I lingered behind and again seated ourselves. "Now, Miss Mary, go ahead and do your worst", I said. She smiled and said—"No, I will try to do my best". She got up—"I'll go for a paper and pencil." "No, don't go, I have a piece of paper right here and my fountain pen", and took out of my wallet a prescription blank of blue paper with my name and address printed at the top. In order that the printing should not be confusing, I tore the paper in two and put the printing aside. Miss A. turned her back and I wrote "What will not curiosity do?", a quotation from Goethe which we had translated from the German at college. I folded the paper and Miss A. turned around and I then followed the instructions so minutely detailed by Dr. Mitchell. When she came to the end, she looked up and said slowly—"What will not curiosity do?" I said—"Yes, you have it exactly."

"Oh, did I get it right?" she said, taking the paper from me and unfolding it, and there was the sentence. We rejoined the family in the library.

The next morning at breakfast Mr. T. said—"Now, Mary, I am on my way in town and must leave you. But now one parting word of advice. You must give up these mind reading experiments. You do not know what effect it may have on your subjects and you do not know what effect it may be having on your own mind."

Miss A. laughingly replied—"Well, I can assure you it is not hurting *my* mind!"

On my way back to Philadelphia, I went over the experiment step by step and always came back to the same point. What had become of the scrap of blue paper which I had put aside? I could not remember seeing it on the table when we left, it apparently was not on the floor, I had not picked it up. The answer was Miss A. herself had picked it up and if so, why had she done so? Here then was the key to the experiment.

Some time later, I was staying over a Sunday in New York and had been invited to Mr. A.'s home for lunch. At the lunch table we were discussing what we would do in the afternoon. Miss A.'s sister had an engagement and would be gone all afternoon. Mr. A. said he owed his doctor five dollars for telling him that morning that he must stay in the house all day because he had a cold. Miss Mary A. and I decided to go for a walk in the Bronx Park. We were crossing a little rustic bridge over a brook. I stopped in the middle of the bridge and leaned against the railing. "Now will you confess that you were unfair to Dr. Mitchell that night at the Jekyl Island Club?"

"Unfair to Dr. Mitchell! What do you mean?" Her face flushed crimson. "And what about the scrap of blue paper that night at the T.'s?"

"I know—I know! When I saw you take that blue prescription blank out of your pocket I knew I was lost. There was no possible way to get a piece of blue paper. But when I saw you tear it in two and put half of it aside on the table my hopes rose and I felt the day might be saved. If I could only get that piece of paper without you noticing it!"

"Well, you succeeded, anyway. Now promise me that you will write to Dr. Mitchell and explain exactly what you did. He will take it all in good part and will be glad to learn the details of your technique".

"Oh, no! I could never do that; I'm afraid I haven't the courage."

"Very well then, the next time I see him I will tell him that you have been placed on the defensive and that your defense has broken down. But what about the light? Dr. Mitchell told us a swinging lantern hung from the roof of the veranda but the light was so dim you could not have read the sentence even if it had been held before you."

"Dr. Mitchell was wrong about that. The door of the club house was open and light from inside made a path of light across the piazza. I took the precaution of seating myself in this path of light."

The next time I was with Dr. Mitchell, I told him about the blue paper episode and my subsequent visit at the A. home in New York. He was forced to smile when he was reminded of his comment at the close of his address in Washington. "Well, I am glad to know the answer. I admit I was much puzzled by it and could not help wondering how she carried out her experiment so successfully."

There is really nothing difficult in carrying out the supposed test. Many years ago I often did it. The only difficult thing about it was to convince one's subject that the whole thing was pure deception from start to finish. Of course, Herrmann, Thurston, Houdini and others of the professional group, as well as Sir Arthur Conan Doyle, Sir Oliver Lodge, and others, were familiar with this and similar experiments.

THE SELECTION OF MADEIRAS FOR A BANQUET

One evening we were seated around the dining room table. Mrs. Mitchell had invited several friends to dinner. But Dr. Mitchell and his brother-in-law, Mr. Richard Cadwalader, had been assigned a very important duty. A banquet for men was to be held in Philadelphia—a very special and a very distinguished banquet, and Dr. Mitchell and Mr. Cadwalader had been asked to select the finest of wines for the occasion. Mr. Hartman Kuhn had volunteered to send samples of Madeira from his famous wine cellar.

Before dessert was served, the butler and footman removed the candelabra and silver ornaments from the table and removed the table cloth. The candelabra and ornaments were replaced on the bare mahogany table and dessert was served. After we had finished, the ladies withdrew to Mrs. Mitchell's library for coffee, leaving Dr. Mitchell, Mr. Cadwalader and myself at the table for coffee and cigars, but—most important—the sampling of the wine, which for me was a most unique occasion.

The butler then came in with a tray containing the samples of Madeira. There were four-ounce bottles, corked, each bearing a number, twenty bottles in all. A glass vessel, Monteith,* filled with water was placed before each of us. The butler placed a writing tablet and pencil beside Dr. Mitchell. He then brought bottle No. 1 from the sideboard and placed it before Dr. Mitchell, who removed the cork, filled his wine glass, passed the bottle to Mr. Cadwalader and then to me.

The glass was then held up to the light to examine the wine. Then the aroma and then a sip of the wine. The glass was placed on the table for a moment or two, then a second sip and the glass put down. A pause.

"Dick, that's the South side of the island."

"Yes, Weir, the South side."

"About 1840."

"Yes, about 1840, '41, '42, along there."

Dr. Mitchell then wrote No. 1 on the pad with the notations.

Specimen No. 2 was produced. After washing our wineglasses in the vessel of water, they were again filled and the solemn performance was repeated. After a pause—

"Weir, that wine has been fined."

"Yes, Dick. That one certainly cannot qualify".

I asked—"What do you mean, Dr. Mitchell, when you say that a wine has been fined?"

Dr. Mitchell put down his glass, turned and

* A letter from Mrs. Helen Sprackling informs me that the Monteith, first described in the late 17th century, was originally a large bowl-like vessel filled with water in which a number of wine glasses, inverted, could be kept cool. Towards the end of the 18th century these were replaced by the Monteith for individual use, which now might include that here described.

stared at me in astonishment—"Why, Elmer, don't you know?"

"No, but I would like to know."

"It means that some time in the history of this wine, milk has been added to it to throw down the sediment—to clear the wine. But the flavor has been changed—the wine can never be the same."

And so on through several samples—the date of vintage, the North side, the South side of the island and brief comments.

"Weir, this wine is gone."

"Yes, Dick, this is certainly gone and no need to comment further."

"What do you mean, Dr. Mitchell, when you say a wine is gone?"

Again, Dr. Mitchell put down his glass, turned and stared at me—"Why Elmer, don't you know?"

"No, I do not know but would like to find out".

"It means that the wine is over 80, it is over-aged, it has lost its flavor—it is gone!"

And so we went on—1832, 1860, 1845—I was beginning to grow dizzy by the time we got up to sample No. 15 or thereabouts. The table was turning around and, as I reached for the next specimen, my cuff caught the rim of my wine glass and overturned it on the bare table with a tinkling sound. For the third time, Dr. Mitchell put down his glass and turned and stared at me—"Elmer, we will have to have you taken home in a cab. You are not used to these parties." There was nothing I could answer to that.

Finally, No. 20 was produced and duly sampled.

The butler, who had stood most patiently behind Dr. Mitchell's chair during this long procedure—the footman having long since disappeared—then placed a sealed envelope on the table. Dr. Mitchell opened it and compared the key with the list which they had prepared. The year of the vintage, the location of the vineyards were almost identical—the year of vintage varying perhaps a year or two. The quality or lack of quality of the wine was duly appraised and two or three of the samples selected as being the finest for the banquet.

The hour had grown late and we went to the library.

For me, it was the most amazing demonstration of skill and long experience in the training of sense perception. Perhaps by now, it has become a lost art.

RATTLESNAKES

In one of my evenings with Dr. Mitchell, I brought up the subject of poisonous serpents, saying that his experience and study of them had been so remarkable and unusual. I spoke of our experiences as students of the Princeton Geological Expedition into the Bad Lands of South Dakota in the summer of 1893. We had no fear of the Sioux Indians nor of the Bannocks—who, by the way, were very treacherous—but we did stand in awe of the rattlesnakes. We sometimes slept in our tents—sometimes on the ground in the open. The nights were cold at 5000 feet, the days intensely hot, 125° in the shade. The rattlesnakes would come into our tents at night. George Swain was sleeping one night with a rattlesnake coiled up close beside him for the sake of warmth. When George awoke he rolled away very carefully and slowly.

One day our cook was lying in his tent reading, his shoes off. He felt something sliding over his foot. He looked down and saw a large rattlesnake crawling over his foot. He lay very still. The snake went out of the tent, across to the camp stove. The oven door was open, the stove was still warm, the snake crawled inside, the cook shut the door and the snake was killed with chloroform.

Late one afternoon, having lost my way, I dismounted, threw the reins to the ground and climbed a butte to look out over the desolate landscape and try to decide upon the most likely location of our camp. Coming down the steep side of the butte, slipping and sliding on the loose limestone, I saw my foot coming directly upon a rattlesnake. We were well protected with heavy high shoes and leggings but our hands were not protected. I could not

stop but it seemed for a moment as though my neart had stopped. It turned out to be an empty skin which a snake had shed and gone on his way, but it looked very real in the half light. And, one day, when I was sitting on the ground busily attempting to dissect the poison sacs out of the head of a rattlesnake, Professor Scott stopped beside me and looked down—"What are you doing, Elmer?" "I am trying to dissect the poison glands out of the jaws of this rattlesnake's head."

"Well, don't do it. Throw the head away and throw your pocket knife away too. If you should cut yourself with it, it would probably kill you." I threw away the snake's head but I did not throw away my knife but scoured the blade carefully with wet sand.

"Dr. Mitchell, you have had a remarkable experience in your study of snake poisons and your 'intermittent ligature' must be known in every country throughout the world. Were you ever bitten by a rattlesnake?"

"Good heavens, no! Of course not. But, I did have quite a startling experience. I kept the snakes in the cellar. One afternoon I had some of them brought up for some study or investigation I was doing at the time. They were in a wooden box or cage. The sides were made of chicken wire. Late that night I was carrying the box down the cellar stairs, holding it in front of me. It was only a few inches from my face. One of the rattlers struck viciously at my face. Fortunately I was protected by the wire screen, but it did give me quite a scare and I was glad to get down to the cellar and put the box down."

DR. MITCHELL IS ADVISED TO CONSULT DR. MITCHELL

After Dr. and Mrs. Mitchell returned from a visit to Germany, Mrs. Mitchell was relating some of their experiences while traveling on the Continent. "When Dr. Mitchell comes in for tea, ask him to tell you about his visit to the great German specialist." Dr. Mitchell came in. "Weir, tell about your consulting the great German professor."

"Yes, I was quite taken by surprise. While we were in Germany, I thought it a good opportunity to get an opinion from one of Germany's most eminent authorities on the nervous system and asked his secretary for an appointment. I was shown into his consulting room and he received me in a most dignified manner and asked me to be seated. I gave him a brief history of my difficulty—said my hand was shaking continuously in spite of every effort to control it. It made writing very difficult and the condition seemed to be growing worse. I would be very grateful for his opinion and advice. He listened attentively and thought for a minute or two after I ceased speaking.

" 'I see you are an American. The best advice I can give you is this. When you return to your country, go to Philadelphia and consult your most distinguished authority on nervous diseases, Dr. Weir Mitchell.'

" 'But I am Dr. Mitchell' ".

"Then followed most cordial greetings and we were invited to come to his home for dinner. Mrs. Mitchell and I went for dinner at the professor's home and the occasion was celebrated over a bottle of champagne."

"WHAT IS THE ANSWER? NO ONE KNOWS."

Dr. Mitchell's office was a bright, cheerful room. On pleasant mornings the sunshine streamed in through the big windows and Dr. Mitchell liked to work in cheerful surroundings.

I happened to go in one morning and although it was a bright, sunny morning, the room was in shadow—a lighted lamp was on Dr. Mitchell's desk. Noting my look of surprise and wonderment, he said—"See what they are doing to me! The Racquet Club is putting up this big building on South 16th Street just beyond my lot and now it has gotten up so high it has shut out all of my sunlight and it leaves me in this gloom. I am obliged to work here every morning with the lamp lighted on my desk. And, the discouraging part of it is that it must always be like this."

Anyone who knew Dr. Mitchell well—who

knew of his fondness for life out of doors—in the open air—in sunlight—salmon fishing in the streams of Canada—realized what it meant to him to have all sunlight shut out of his consulting room where he spent his daily morning working hours.

I sat down in a chair beside his desk and was looking at four pink roses in a vase on his desk, admiring their delicate coloring. Then every petal at the same instant fell onto the polished surface of the desk, leaving only the bare stalks. "It seems odd", he said, "that every petal should let go at the same time. There was no perceptible current of air stirring in the room. It is like a great tree standing in a forest which has withstood the strains of storms and tempests for a century and more. And then, on a quiet calm day when there is not even the slightest trace of a breeze stirring in its branches, it comes crashing down. What is the answer? No one knows."

BAR HARBOR

Looking back across the years, I recall, too, many happy and stimulating hours spent at "Dolce Far Niente," the Mitchell summer home at Bar Harbor, which stood on the rocky shore of Frenchman's Bay, looking out to sea.

Dr. Mitchell had planned to read a poem he had written. In the narrative of the poem the music of a violin was heard from time to time. Mrs. Mitchell sent out invitations for tea at five o'clock. The curtains were drawn, there were lighted candles on the piano, the guests began to arrive; among them, Albert Spalding with his violin, to provide the musical interludes during the reading of the poem.

Dr. Mitchell came over and stood beside the piano, his features shown clearly in the light of the candles; Albert Spalding arose and took his position—the audience grew silent. After the reading of the first stanza and the music of Spalding's violin, there was a ripple of applause. Dr. Mitchell very gravely and very politely asked the audience to please refrain from applause until the reading of the poem was concluded. From that point on, the audience sat very still.

When the reading was finished, Dr. Mitchell smiled. There was an outburst of enthusiastic applause and many congratulations for Dr. Mitchell, and also for Mr Spalding. Then tea was served; altogether a very enjoyable party.

Dr. Mitchell was always enthusiastic about Mount Desert—the wooded hills, the trails and bridle paths, the rugged rock-bound coast and the pounding surf—the open sea. Even in his last summer, dressed in sports jacket, knickerbockers, carrying an alpen-stick, he climbed the hills with his younger companions—"Always take very short steps when climbing a mountain," he said. "It saves one's muscles from fatigue—something I learned from the Swiss guides in the Alps."

MEDICAL HISTORY

Dr. Mitchell was well versed in medical history and in our Colonial history, as his writings testify. I well remember his keen interest and enthusiasm when he examined the M.D. diploma conferred on Jonathan Elmer, in 1771, by the Medical School of the College of Philadelphia, now the University of Pennsylvania. This remained in a metal case for a hundred and thirty-five years. It seemed to me that it was just the kind of document, coming down to us from the Colonial period, that would appeal to Dr. Mitchell. I took it to his house one evening and he received me in his office.

"What have you in that mysterious-looking cylindrical case?"

I took out the diploma and handed it to him. He unrolled it on his desk. His face lighted up with enthusiasm as he glanced over it—"There's John Morgan", he exclaimed; "There's Benjamin Rush—There's William Shippen—Adam Kuhn—Kinnersley"—and the other signatures, pointing to each in turn; and then he carefully examined the diploma. He was familiar with the personal history of the members of the faculty and with the early years of the first medical school in the Colonies. This was the first diploma granting the degree of Doctor of Medicine to students of the original class which began the study of med-

icine in this country. And now, it appears, this is the only one in existence.*

Dr. Robert Abbe, of New York, was an admirer of Dr. Benjamin Rush—a student of his life and writings. He came over to Philadelphia to attend a meeting of the College of Physicians and to present to the College the gold watch of Benjamin Rush which Dr. Rush had carried through the great epidemic of yellow fever in Philadelphia in 1793. Dr. Abbe was the guest of Dr. Mitchell. I had had the pleasure of knowing him at Mount Desert, where he spent his summer vacations.

Toward the close of the meeting, I asked Dr. Mitchell and Dr. Abbe if they would like to stop at my office on their way back to Dr. Mitchell's house, in order that Dr. Abbe might have the opportunity to see the diploma and examine the signature of Dr. Rush, as well as the signatures of the other members of the faculty of the first medical school in America. They said they would come. A little later, in Dr. Mitchell's brougham, coachman on the box and the handsome pair of horses, they arrived at my office.

The framed diploma hung over the mantlepiece. Dr. Abbe began reading it, and then studied the signatures, that of Dr. Rush especially. They were all then sharp and clear, written with quill pens, of course. Dr. Mitchell, meanwhile, walked about the room—looking at pictures, framed letters, books, a sperm oil lamp of Colonial days—various odds and ends. As they were leaving, I went out with them to the carriage; and when they drove away, I had a feeling that I had just said good-night to two very important visitors.

THE LAST VISIT

One evening late in December, 1913, Mrs. Mitchell was having a small dinner party. We were talking in the library, waiting for one or two guests to arrive, Dr. Mitchell came into the room, immaculately dressed as usual—Tuxedo suit, an embroidered waistcoat, bright-colored fancy silk socks and patent leather pumps, and, I think, a carnation in his lapel. He greeted us in his usual cordial, cheerful way and then his daughter-in-law, Mrs. John K. Mitchell, came in, and after the greetings Dr. Mitchell took her hand and said —"Just think, Anne! 'Westways' has sold thirty thousand copies and it has only been out two weeks!" He seemed so pleased, and so were we all pleased, even though we had not read "Westways." It was a pleasant little dinner party—each one in such good spirits and no thought that it was the last. Dr. Mitchell, who was eighty-four, was taken ill within a week and died of pneumonia. Mrs. Mitchell contracted pneumonia and died about a week later.

For me, it ended twenty years of the most enjoyable and congenial friendship one could possibly know. Over my desk hangs a large framed etching of Dr. Mitchell—a most lifelike likeness—a fine work of art, and at the bottom Dr. Mitchell has written in his shaky handwriting—

Walter G. Elmer from his friend
and his father's friend
Weir Mitchell

These recollections have been set down from memory—no diary—no notes—no memoranda. However, the memory of these various episodes is clear and vivid.

VESPERAL

I know the night is near at hand.
The mists lie low on hill and bay,
The autumn sheaves are dewless, dry;
But I have had the day.

Yes, I have had, dear Lord, the day;
When at Thy call I have the night,
Brief be the twilight as I pass
From light to dark, from dark to light.

October 1899 S. Weir Mitchell

* Dr. Walter G. Elmer recently presented the diploma to the University of Pennsylvania. [Ed.]

Memoir of Ralph Shepard Bromer (1886–1957)*

By PAUL A. BISHOP, M.D.

RALPH SHEPARD BROMER, a Fellow of the College, died in the Bryn Mawr Hospital on September 25, 1957, some twelve hours after a cerebral hemorrhage. He had been in good health and was busy planning to attend the annual meeting of the American Roentgen Ray Society in Washington the week following. He had been anticipating the great pleasure of another reunion with his many friends.

Dr. Bromer was born in Schwenksville, Pennsylvania, seventy-one years ago. He received his preliminary education at the Penn Charter School in Philadelphia. He graduated from Yale University with a Bachelor of Arts degree in 1908 and from the Medical School of the University of Pennsylvania in 1912. After a two year internship at the Pennsylvania Hospital he served as a House Officer in the Children's Hospital of Boston. Together with Dr. Astley P. C. Ashhurst, then Professor of Surgery at the University of Pennsylvania, he made an extensive tour of the leading orthopedic clinics of Europe. This trip was cut short by the outbreak of the first world war. The attraction and admiration between these two men was cemented by his trip, however, and increased through the many years they worked together. Dr. Ashhurst exerted a great influence on the future career of Dr. Bromer.

In spite of the war Dr. Bromer remained in Paris for several months, working in the American Hospital, before returning to begin practice of orthopedics in Louisville, Kentucky. In 1917, at the urging of Dr. Ashhurst, he came back to Philadelphia to take charge of the Department of Roentgenology at the Episcopal Hospital. Three months later he entered active military service and was sent to the Army School of Radiology in New York. Subsequently, in France, he served with distinction as Radiologist and Adjutant of Base Hospital 34.

On his return in 1919 he resumed his duties at the Episcopal Hospital and shortly thereafter accepted appointments as Roentgenologist at the Orthopedic and Children's Hospitals. In these three positions, which he held simultaneously for several years (thirty years at the Children's Hospital), his early interest and training in orthopedics and in diseases of children came to the fore. He had a phenomenal memory for the details of rare cases he had seen or read about and he was soon recognized as the local and a national authority on diseases of bone. It was to him that all local roentgenologists and many from a distance came with their stacks of films of mystifying cases. We always came away rewarded and impressed by that unselfish spirit that was so typical of him. His publications and lectures on rickets, scurvy and lues of bone are classic. He was able to bring clarity and order to the confusion of pathological changes and diagnostic signs that were reported in the literature on these complex diseases.

In 1932 Dr. Bromer became the Director of the Department of Radiology at the Bryn Mawr Hospital, a position he occupied with distinction until he retired, due to ill health, only a short time before his death. His contributions were an important factor in the medical and physical growth of that institution. He served as president of the Medical Staff and as an influential member of its Executive Committee. A tireless worker with great intellectual honesty, and, I might add, a marvelous sense of humor, his department became a center of medical activity.

He was an active teacher in the Medical School of the University of Pennsylvania and

* Read before the College of Physicians of Philadelphia, 5 March 1958. Prepared and published at the request of the Council of the College of Physicians of Philadelphia.

at the time of his retirement was made Emeritus Professor of Clinical Radiology in its Graduate School. His greatest contribution as a teacher, however, was in his daily contacts with interns, residents and young radiologists. He maintained a youthful attitude toward them and was never too busy to enter into their problems with enthusiasm and understanding. He was loved by them all. John Mason Brown recently said, "All persons are contemporaries if only their interests overlap." Perhaps that was his secret.

He played an important role in the activities of radiological societies. He was a past president of the Philadelphia Roentgen Ray Society and of the American Roentgen Ray Society. He served on the Executive Council of the latter on several occasions and took an active interest in its *Journal*. He was a member of the American Board of Radiology and served as an examiner of this Board for twenty years.

Ralph Bromer was a scholar, a gentleman with many cultural interests and a sound appreciation of music and the arts. His library contained an exhaustive collection on Napoleon and on the Civil War. He was an avid gardener and, by the Lord's grace, it was in the garden he loved so well that his final illness overtook him.

He is survived by his wife, Alice Rupp Bromer, a son, Ralph Shepard Bromer, Jr., and a daughter, Catherine Bromer Wren.

In the death of Ralph Bromer, medicine, and especially radiology, has lost a leader of distinction. Those of us who were so fortunate as to know him and work with him have lost an inspiring friend and a wise counselor.

Memoir of George Wagoner (1896–1957)*

By BEN TERTIUS BELL, M.D.

OUR distinguished Fellow, George Wagoner, orthopedic surgeon, research scientist, teacher, administrator, soldier and sportsman, died suddenly on April 29, 1957 near Ligonier, Pennsylvania.

The surgeon began his training under his father, Dr. George W. Wagoner, a leading general practitioner of Johnstown, Pennsylvania. After his college years at the University of Pennsylvania, he was graduated from its Medical School in 1922. He completed his internship at the Philadelphia General Hospital in 1924, after work not without romance, for he married Marjorie S. Jefferies, M.D., a fellow intern at venerable Blockley. His training continued at the University of Pennsylvania as the first John Hunter Fellow in Research Surgery (1924–1926), and at the University of Innsbruck (1929 and 1934) under Professor Lang in Bone Pathology. He began active practice of surgery at Bryn Mawr Hospital in 1926 and reached the rank of Chief of the Orthopedic Service. At the time of his death, he was Attending Orthopedic Surgeon at the Graduate Hospital of the University of Pennsylvania and consultant in this specialty at Pottstown Hospital and Woman's Hospital of Philadelphia.

Dr. Wagoner's work as a research scientist began with distinction in medical school, where he devoted much time to work with Drs. A. N. Richards and Joshua E. Sweet. Dr. Wagoner developed then an original method of measuring blood flow through application of the Venturi meter principle, for which he was elected to membership in Sigma Xi. Under the guidance of Dr. Sweet, he developed surgical techniques on animals and formed a deep and lifelong friendship with his beloved preceptor. As Professor of Orthopedic Research and Director of its Laboratory at the Graduate School of Medicine of the University of Pennsylvania, he introduced the celloidin mount technique for microscopic examination of total specimen sections, a method which is now used by leading bone pathologists and which yields beautiful material for demonstration purposes. He built thus a large collection of pathologic material. In 1936–37 he studied the relationship of climate to the epidemic of poliomyelitis for the National Foundation for Infantile Paralysis.

As teacher, Dr. Wagoner drew on his clinical experience and his superb microscopic slide collection for a well-rounded course in Orthopedic Pathology at the Graduate School. In this course, he gave terse, well-documented lectures but encouraged mainly the study of the slides, uninhibited informal discussion and argument—with stimulating results.

The many honors bestowed on Dr. Wagoner brought with them the duties of administrator. He served as Secretary-Treasurer of the Staff of Bryn Mawr Hospital from 1947–1950 and as its President from 1950–1951. In addition, he spent many years on the Executive Committee of this institution. His ability as a keen parliamentarian was well-recognized and staff members were grateful for the aplomb with which he conducted the all-too-many meetings. It was at one of these meetings, not long before Dr. Wagoner's passing, that he defined the duty of the physician to the community not only as a healer of man but also as a symbol of morality.

Much more must be said of this Philadelphia physician who served his community and country in so many ways. Dr. Wagoner was active in both World Wars. In the first, he served in the United States Ambulance Corps; and in the second, as a Commander in the United States Naval Reserve. He was attached to the Marine Corps at Parris Island and saw sea duty on the U.S.S. Pinckney assigned to

* Prepared and published at the request of the Council of the College of Physicians of Philadelphia.

the Pacific Theater. His interest in military affairs and the security of his country continued into the Post-War period when he became Director of the Medical and Health Division of Montgomery County Civil Defense, and co-chairman of the Advisory Committee to the Selective Service System of Montgomery County.

Dr. Wagoner had many responsibilities and interests beyond his specialty. He was a devoted father and family head. His enthusiasms included American history, photography, painting, carpentry and cabinet making. His career as sportsman included playing varsity football for the University of Pennsylvania. Big game hunting, including bears and alligators, and fishing were the favorite sports of his later years. Dr. Wagoner was known for his dash and fearlessness in the football years and these qualities stayed with him to the moment of his death. The memory of his honesty and brevity with his patients, his friendly bark in the operating room, his unswerving devotion to his causes will remain in the hearts of those who knew him. He will continue to live in these memories, through his writings, through his two daughters, (one of whom is yet another Philadelphia physician), and through the monument of his life-long labor, orthopedic surgery. Dr. Wagoner was a member of the following societies:

American Medical Association
Halstead Society
College of Physicians of Philadelphia
Philadelphia Orthopaedic Club
Societe Internationale De Chirurgie Orthopedique et De Traumatologie
American Orthopaedic Association
Society of the Sigma Xi
American Historical Association
Pennsylvania Academy of Science
Pennsylvania Historical Society
Rolling Rock Club
Boone and Crockett Club

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Memoir of William Egbert Robertson (1869–1956)*

By RICHARD A. KERN, M.D.

WILLIAM EGBERT ROBERTSON was born on July 1, 1869, in Camden, N. J., the son of William Robertson, who was of Scottish origin, and Ellen (Harding) Robertson. At the age of 17 he began three years of premedical study in the Biology Department of the University of Pennsylvania, and then entered its School of Medicine from which he was graduated in 1892. After a few months of internship in the Episcopal Hospital, Philadelphia, and the Cooper Hospital, Camden, he went abroad for a year of study in Heidelberg and Vienna, where he devoted himself to internal medicine and to gross and microscopic pathology. In 1893, he returned to Philadelphia where he spent his whole career as physician and teacher.

From 1893 to 1896, he served as Instructor in Medicine and Assistant to Dr. M. Howard Fussell in the Out-patient Department of the Hospital of the University of Pennsylvania. In 1897, he became Pathologist to the Episcopal Hospital, a position which he held until 1904, when he became Visiting Physician to that hospital, and so continued for 27 years until 1931.

In 1897, he also became attached to the Medico-Chirurgical College and Hospital, first as Instructor of Bacteriology and Microscopic Pathology, later becoming Instructor in Physical Diagnosis, then Instructor in Medicine, and eventually Assistant Professor of Clinical Medicine.

When "Medico-Chi", as it was familiarly known, merged in 1916 with the University of Pennsylvania to become its Graduate School of Medicine, Dr. Robertson became Professor of Medicine and Head of the Department in the School of Medicine of Temple University and Visiting Physician to its Samaritan Hospital. These positions he held until 1933 when he was made Professor Emeritus.

He also became Physician to the Philadelphia General Hospital and served for a time as President of its Staff. Other hospitals in which he was Visiting Physician were the Garretson Hospital and St. Luke's and Children's Hospital Medical Center, and he was Consultant to the Rush Hospital, the Kensington Hospital for Women, and the Northeastern Hospital.

A member of the American Medical Association and its component bodies, Dr. Robertson took an active part in the affairs of the Philadelphia County Medical Society, serving on many of its committees, and its Board of Directors. He was President of the Society in 1937–38, and finally a member of its Board of Censors. For a number of years he was a member of the State Health Committee of the National Youth Administration.

In 1903 he became a Fellow of this College and then began an era of sustained interest and participation in the affairs of the College, particularly of the Library, that continued to the end of his life. From 1934 to 1955, he was a member, elected or *ex officio*, of the Library Committee, and from 1940 to 1944 served as its Chairman and Member of Council. In 1945 he was elected Honorary Librarian, a position that he held until he asked not to be reelected in December, 1955, only 8 months before his death. He took an active part in the work of the Sections on General Medicine and on Medical History, and was Chairman of the former in 1927 and 1928, and of the latter in 1934 and 1935. He served on the Alvarenga

* Read before the College of Physicians of Philadelphia, 2 April 1958. Prepared and published at the request of the Council of the College of Physicians of Philadelphia.

Prize Committee and on the Committee on Industrial Medicine and Public Health.

He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He held membership in the American Association for the Advancement of Science, the Society of the Sigma Xi (Pennsylvania Chapter), and the Pathological Society of Philadelphia. An avid student of Medical Journals, he associated himself in 1908 with a group of contemporary physicians of this city, including Drs. Theodore H. Weisenburg, George E. Pfahler, Claude P. Brown, Francis A. Faught, William E. Krewson, J. Torrance Rugh, and Gordon Saxon, in the newly organized Medical Literature Club, which continued to meet monthly during the academic year until 1947. He was also a member of the Union League of Philadelphia and of the St. Andrew's Society of Philadelphia.

Dr. Robertson was a scholarly man, with a consuming interest in history. When asked in a questionnaire about his hobbies, he gave just one: "Reading history: mediaeval and general." His conversation as well as his writings was adorned with instructive references to the past. Medical history received his special attention and it was he who brought the distinguished medical historian, Dr. Victor Robinson, to the School of Medicine of Temple University as Professor of History of Medicine. One of Dr. Robertson's last publications, written in 1948 at the age of 79, was a biographical study on Michael Servetus, medical martyr and discoverer of the pulmonary circulation, which appeared in the Victor Robinson Memorial Volume.

Dr. Robertson in his long career contributed many articles to medical journals, also the chapter on diseases of the nervous system for a Textbook of Medicine. With his son, Dr. Harold F. Robertson, he wrote a textbook entitled "Diagnostic Signs, Reflexes and Syndromes," that has gone through several editions.

On April 26, 1893, he married Anna Gertrude Frederick, who departed this life on December 14, 1948. They had two sons: Harold Frederick and Ralph Egbert. The latter died in October, 1955.

On December 14, 1949, the first anniversary of his wife's death, Dr. Robertson suffered a hemiplegia that made him a complete invalid for the rest of his life. He died on March 9, 1956, in his 87th year.

These are the essential data of his curriculum vitae. While they indicate his work and his interests, they tell nothing of the man himself: his charm of personality, his grace of manner, his steadfastness of purpose, the deliberate thoroughness with which he did things, his meticulous attention to detail. He was soft-spoken and courteous, patient and forbearing, but let no one think that his mildness of manner betokened any weakness of resolution or vacillation in action.

It is told of him that many years ago in one of the hospitals to which he was attached he made a diagnosis of intestinal perforation in a patient with typhoid fever. A surgeon who was called in consultation did not agree with this diagnosis and declined to perform an immediate operation. "Very well," said Dr. Robertson, "if you won't, I will." In a sideroom of the medical ward he opened the patient's abdomen, found the perforation and inserted a Paul tube: the patient recovered. Truly, it could be said of him, "suaviter in modo, fortiter in re."

As a teacher he was hampered by his zeal for perfection, in that he must needs present every detail of what was known of a subject, so that lecture hours were often too short and too few to give him full scope. But this very quality of thoroughness made him an excellent consultant, much sought after by his colleagues.

His devotion to the Library of this College received increasing indulgence in the years after his retirement from teaching and hospital practice. He endeared himself to our staff by his ever-willing helpfulness and service. I close this memoir with the eloquent tribute paid to Dr. Robertson by one who had served with him

over the years, our Curator of Library Historical Collections, erstwhile Librarian, Dr. Walton B. McDaniel, 2d, in the Annual Report of the Library Committee last year: "One who served with him records in gratitude and an understandable sense of loss, the memory of his faithful discharge of his responsibilities, his progressive intellectual outlook, based on wide reading and scholarly interests, his sense of humor, and not least, his sympathetic understanding of human problems. A Fellow charged with the responsibilities that were his may discharge them effectively in any number of ways, of course. Dr. Robertson's was a modest, unassertive way whose effectiveness—unfortunately for historical purposes—is less likely to show up in the minutes than in the memories of those whose actions were subtly influenced by the example he set."

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Health and Hospitals for An Aging Population[1]

By LEROY E. BURNEY, M.D., M.P.H.[2]

IT WAS never my privilege to meet Dr. Anders whose life and work are commemorated by this occasion. When the College of Physicians of Philadelphia honored me by an invitation to deliver the James M. Anders Lecture, I wished I had known that beloved physician, teacher, and community leader as his colleagues had known him. The formal biographies and his published works could not provide such an acquaintance, but I found the perfect introduction in a rare little volume on the shelves of our National Library of Medicine, now a treasured unit of the U. S. Public Health Service.

Many of you will recall the publication. It consists of the collected addresses delivered at the testimonial dinner tendered to Dr. Anders, November 29, 1927, on the occasion of his Golden Anniversary as a doctor of medicine in the city of Philadelphia. (1) A few hours with that volume were well spent. I felt that I had known Dr. Anders intimately. I felt the large respect and trust he inspired in his students and colleagues. In the end, I felt a kind of awe. For thirty years ago, that wise leader had seen, far beyond this evening's point in time, the future shape of medicine and public health.

[1] James M. Anders Lecture XXXVII, College of Physicians of Philadelphia, 2 April 1958.

[2] Surgeon General, Public Health Service, U. S. Department of Health, Education and Welfare, Washington 25, D. C.

Let me give you' two examples from Dr Anders' own valedictory address.

"My earnest hope", he said, "is that I may live to see Philadelphia medicine carried to a higher pitch in the domain of pure original research . . . an objective possible only by a united cooperative effort of all medical Philadelphia." In Dr. Anders' remaining years, there had been progress toward that objective; but even a man of his vision could scarcely have imagined the scope, volume, and quality of medical research in Philadelphia's institutions today. This city has become one of the great medical centers of the world, and its future in research is assured.

Again, Dr. Anders said: "In the future, the medical profession will strive to prevent disease and to secure adequate means to meet sanitary necessities since (the profession) will be composed of *preventive* rather than curative physicians."

This prophetic sentence provides an admirable text for my discussion this evening. Health and hospitals for an aging population comprise a problem which confronts every state and local community in the United States. Its solution depends primarily upon the realization of the three-fold goal set by Dr. Anders for the medical profession: namely, "to prevent disease, and to secure adequate means

to meet sanitary necessities", and to become "preventive rather than curative physicians."

The challenge to physicians is explicit. The problem, however, is not exclusively a medical problem. The economic and social components are large and they influence—sometimes actually create—the medical components which are the physician's direct responsibility. The costs of hospitalization, for example, may limit the physician's efforts to render the type of medical care which he knows is essential for the well-being of a particular patient. In long-term illness especially, he may have to be content with incomplete care because the costs of hospitalization cannot be met.

The current status of medical science also influences the physician's capacity to solve the problems of disease. Unsolved medical problems have a tremendous impact on the Nation's social and economic efficiency. We have not effectively controlled the acute upper respiratory diseases, for example. It is an impressive fact that during 30 weeks in the first wave of the Asian influenza epidemic (June 30, 1957 to January 25, 1958), acute upper respiratory diseases accounted for more than 435 million days of illness in bed. This total is equivalent to 1,200,000 person-years lost from usual activities such as work, housekeeping, or school. Even though the medical and public health professions were fully prepared in advance to deal with the threat of a widespread, severe influenza epidemic, we could not avert the total impact of this group of diseases.

With your indulgence, I will discuss some of the socio-economic factors encompassed by my subject, with special reference to present and potential opportunities for meeting the changing health problems of our times.

Changing Characteristics of the Population

Rapid change is the outstanding characteristic of the American scene in this second half of the twentieth century. It is the primary factor which affects the Nation's overall problem of health and hospitals and which strengthens the inter-relation of medical, social, and economic efficiency. Population trends are basic to the factor of change, and understanding of medical and health problems is dependent on understanding of the characteristics of our changing population.

The United States is in a long-range period of large population growth. From 1940–1957, the population increased by nearly 31 per cent, from 130 million to over 170 million, with very little addition from immigration. As recently as 1946, demographers predicted that the country would reach a peak of 165 million along about 1990. Today, they are predicting anywhere from 210 to 228½ million in 1975—and this on the conservative assumption that there will be no improvement in mortality after 1960. (2)

We have also a population of extraordinary mobility. It is not only the long-range move from the farm to urban areas. It is also a vast criss-cross mobility. People within the same state are moving from the small towns to the city, and from the central cities to the suburban fringes of metropolitan areas. The character of the central city population has changed, with substantial losses of young middle-class families to the fringe suburbs and influxes of low-income families from instate and outstate into abandoned neighborhoods. It has been estimated that one in every five Americans changed their residence in any given year of the period 1940 to 1950.

The "baby boom" which began in the mid-forties continues apace. Since 1954, there have been more than 4 million live births annually through 1957. Families are larger, with increases since 1945 in the numbers of third, fourth, fifth, and even sixth births. Marriages are younger, with the median age at first marriage of 20 years in females. The birth rate has been at, or higher than, 25 per 1000 population since 1952.

The mortality experience of the United States has been the most dynamic factor in the so-called "aging" of the population. The crude death rate in 1900 was 17.2 per 1000 population, and in 1957 it was estimated at 9.6 per

1000. (3) The principal reductions have been in rates due to infectious diseases, and in infant mortality. The provisional rate of 1957 represents a rise of about 2 per cent over 1956. A comparable increase was observed in 1956 over 1955. It is too early to predict that these increases represent a reversal of the long downward trend in mortality. They do, however, highlight the difficulties in making a substantial impact on deaths due to chronic diseases, congenital malformations, and accidents.

Life expectancy at birth has increased for males from 48 years in 1900 to 66 years in 1950; and for females, from 51 to 72 years. (4) Thus, there are more women in the population than men, and there has been a corresponding increase in the employment of women. The major increase in recent years has been the employment of older married women, whose children no longer require constant supervision. From the point of view of health and medical care, this means that there are fewer responsible family members available to take over the home care of patients with long-term illness or of the aged.

The prolonging of life has increased the number and proportion of older persons in the population. In the half century 1900 to 1950, the total population doubled, but the number of persons 65 years of age and older increased fourfold. There are now more than 14½ million individuals in the 65-year-and-over bracket, comprising 8.5 per cent of the population.

It has been common in the past two decades to predict a constantly rising ratio and number of older persons in the population. Projections of this type must be considered with caution, since they affect planning for the health and medical care of the total population, especially of the aged. The number of older persons may continue to rise; but unless some striking change takes place in the control of chronic diseases, which contribute the highest mortality rates in the middle-age groups, we must anticipate a slower rate of increase in the older age groups. By 1975 persons entering the middle-age groups, 45 years and over, will be from the generations born in the 1930's when total births were low and when infant mortality and childhood deaths from many infectious diseases were still relatively high. The cohorts arriving at middle and old age, therefore, will be considerably smaller in the last quarter of this century than those now reaching the ages of 45 and over.

Changing Patterns of Illness and Mortality

The changing patterns of illness and mortality since 1900, especially in the past twenty years, have been emphasized by all professional and public groups concerned with health and hospitals. The reduction in the prevalence and mortality of a considerable number of infectious diseases have focused attention on the so-called chronic diseases and mental illness. Because both mortality and disability rates from these causes are highest in adult life and increase progressively with advancing age, there has been increased emphasis on the health problems of aging.

Practicing physicians and administrators of hospitals and health services, however, are obliged to take the total burden of disease into their considerations, giving equal attention to all causes of illness and death and to all groups affected thereby. Age, sex, economic status, and patterns of living, no less than the nature of the disease, are relevant to the health of the individual, to the physician's therapeutic decisions, and to the type of medical facilities required for the care of the patient. The duration of disease also is of signal importance. Advances in diagnosis and therapy have had as striking an effect on this factor as on mortality.

To understand these changes and to plan wisely so that they will be met satisfactorily for the individual patient and for the whole community, we need facts about the burdens of illness in all parts of the country. We need to know the relative impact of acute and of long-term illness; of specific diseases and their incapacitating effects on different population groups.

The National Health Survey, established in

the Public Health Service by Congress in 1956, is now beginning to give us new data on these and other factors. It would appear that the prevalence of chronic conditions is considerably higher in the noninstitutional population than has been estimated in the past. The terms of reference in previous estimates do not permit specific comparisons with the new National Health Survey data. It is significant, however, that a 1950 estimate of total prevalence of chronic diseases and impairments gave a figure of 28 million persons, about one-sixth of the population. In contrast, the National Health Survey finds that nearly 70 million persons, or 41 per cent of the population, reported one or more chronic conditions. (5)

Data never before available for the country as a whole also show that 10 per cent of the people—more than 17 million individuals—are limited in some degree by chronic conditions. Over 4 million are incapacitated to the extent that they cannot perform their main activity, such as attending school, going to work, or keeping house.

The pattern of illness and mortality as revealed by available data, clearly delineates two major areas for concerted action. One is the intensive treatment and restoration of individuals with severely disabling conditions. A large proportion of these are adults in the middle and later years of life; but they include children and youths already severely afflicted by chronic conditions. A survey of mental deficiency recently completed by the National Association for Retarded Children, for example, reports that more than 3 per cent of the infants born in this country each year will never reach a 12-year level of intelligence. (6) With high birth rates and with available techniques for preventing deaths due to acute diseases and congenital malformations, this means that we can anticipate an increasing number of children and adults requiring intensive treatment and rehabilitation, because of this one type of disabling disease alone. The task presented by victims of prolonged disability is essentially a salvage job. It is one that can no longer be shirked, but it does not attack the burden of illness at the roots.

The second major area for concerted action is the prevention of chronic disease. Here the burden is attacked at the roots. The best hope of reducing prolonged severe disability and of averting premature death is in the preventive approach, focusing the attack upon the early and middle years of life. These age groups, with lower rates of morbidity and mortality due to chronic disease, offer the greatest challenge to medicine and public health.

Changes in Medicine and Public Health

Our ability to meet that challenge has been enormously enhanced by changes in the patterns of medical thought and practice. The close of World War II released a freshet of pent-up thought and action into channels of medical and health services for the civilian population. There were numerous distinguished gatherings of professional leaders to provide a focus for the exchange of ideas and a means for disseminating the new emphases.

As early as 1947, four major national organizations affirmed the preventive approach to chronic disease in the following statement:

> "The basic approach to chronic disease must be preventive. Otherwise the problems created by chronic diseases will grow larger with time, and the hope of any substantial decline in their incidence and severity will be postponed for many years." (7)

This Joint Statement by the American Hospital Association, American Medical Association, American Public Health Association, and American Public Welfare Association led to the establishment of the Commission on Chronic Illness which they sponsored throughout its brief and brilliant life, 1949–1956. The first national conference conducted by the Commission in 1951 was on the preventive aspects of chronic disease. This was a bold venture, for the concept that *all* chronic diseases should be subjected to the preventive approach was still a novel one and its realization seemed remote.

In the years since affirmation of the preventive approach to chronic disease, medical terms unheard of in 1947 have entered the common vocabulary. The new techniques they specify have entered the average physician's daily practice and many of them relate to the primary or secondary prevention of chronic illness. A few of these are:

Anticoagulants—steroid compounds—exchange transfusions—chemoprophylaxis of rheumatic fever and subacute bacterial endocarditis—isoniazid—Salk vaccine—vaginal cytology—and tranquilizers.

Such practical achievements as these have brought the medical profession closer to Dr. Anders' goal of being "preventive rather than curative physicians"—whether or not they realize it or consciously seek the preventive approach. The numbers of cardiovascular patients who are surviving for long periods their first coronary attacks under modern medical management testify to this observation. The suppression of rheumatoid inflammation by the use of steroid therapy has helped to ameliorate crippling in rheumatoid arthritis. The identification of Rh blood incompatibility and the use of exchange transfusions in affected infants has reduced the proportion of cerebral palsy cases due to erythroblastosis fetalis from 10 per cent of all cases to 1 per cent. (8) The death rates from rheumatic heart disease and subacute bacterial endocarditis have been declining for many years. Since the introduction and universal use of penicillin, they have dropped sharply in the age groups at special risk. The combined rates from these two heart ailments in 1955 was less than 4 per 100,000 of the population aged 5 to 24 years. (9)

According to physicians who have devoted their long careers to the attack on tuberculosis, isoniazid therapy has actually altered the pathology of the disease. (10) Certainly we in public health know that it has revolutionized the approach to control. It has reduced sharply the average length of stay in sanatoria. It has made possible the treatment of thousands of patients in their own homes by their personal physicians; and it has returned much larger numbers who have received the needed sanatorium care to their physicians for supervision and continued therapy. On the near horizon is the prospect of applying the chemoprophylactic properties of isoniazid broadly. This should prove as effective in tuberculosis control as has the use of penicillin in the prevention of rheumatic fever.

Medicine and public health are just beginning to feel the impact of the world-wide, postwar expansion of medical research. The expansion has been notable in our country. Measured in annual expenditures from all sources, research in the medical and biological sciences has increased fourfold in the past decade, from about $88 million to nearly $360 million in 1957. The public is better informed today on the findings of scientific research than at any time in the past. The people are not content, however, with reports of laboratory and clinical investigations. They expect a return on the Nation's research investment in the form of prompt and effective application of the findings.

It was natural thirty years ago that so astute an observer as the late Dr. David Riesman could see little opportunity for public health action in the field of chronic disease when he was speaking of Dr. Anders' notable contributions to public health work and medical organization. "All the degenerative diseases . . . are difficult to combat in the mass", he said. "They are not communicable, although they are to some extent transmissible by heredity. The attack is an individualistic one rather than civic or national." (11)

With one minor exception, public health would wholeheartedly agree with those observations today. The causes of long-term illness are indeed difficult to combat in the mass. Man's "genetic inheritance" is the subject of the most challenging medical research today relative to an increasing number of chronic conditions. At the present time, the attack upon most of the chronic diseases is still "an individualistic one." Successful application of preventive techniques requires a close relationship between the patient and his personal

physician. It requires also a similar relationship of these two central characters in the drama of illness with other physicians, with nurses, technicians, therapists, educational and vocational counselors, various types of institutions and community organizations.

There is a role for public health in this drama, too. We would alter Dr. Riesman's 1927 observation in only one respect and we are confident that in 1958 he would agree with us. We would say, "The attack is an individualistic one *and* a civic and national one." For it is clear to the professions and the public that the success of the physician and his patient in their mutual attack on chronic illness depends very heavily upon the civic attack. That is to say, upon what the community does in the way of providing the array of facilities and services essential to achieving success. I realize that I am speaking to "the converted." Few cities in our country have gone as far as Philadelphia in carrying out this "civic attack" on chronic illness and the problems of aging; few medical communities are as aware of the civic contribution or as alert to support and accept it as is Philadelphia medicine.

The civic or community attack depends in turn upon the national attack. It would be unrealistic to ignore the fact that no community, no particular group in this country can achieve its purposes unaided by national knowledge and skills, national concern and support. Rest assured I do not mean by "national" the Federal Government. No one could deny the national role of the Government; but it would be equally unrealistic to attribute to it the sum of national wisdom, power, and control.

The national reservoirs of corporate strength, wealth, and production; the reservoirs of learning in the bewildering technology of our times; of skill in the organization of services are distributed not so much geographically as throughout industry, governments, corporate business, and private associations. The individual community perforce turns to one or more of these national resources whether it be to attract industry to a small town that has lost its self-sufficiency or to redevelop a deteriorating central city; whether it be to expand medical research and education or to construct a new public school or nursing home; whether to establish a home care service like Philadelphia's pioneer program or to open a new supermarket in the newest suburban area.

In the local community, the state, and the country as a whole, the application of preventive concepts requires the mobilization of resources; hence also the cooperative action of many diversified public and private organizations. The official public health agencies of today visualize the role of "mobilizer" as their primary contribution to the attack on chronic diseases and prolonged disability. This would involve application of many of the usual activities of health agencies to new problems. Among these, familiar in communicable disease control, one could mention: epidemiologic investigations and community surveys designed to delineate specific needs in the fields of chronic disease and health of the aging; laboratory diagnostic services in communities lacking such facilities; home nursing, nutritional counseling, visiting therapists, and other services to supplement the physician's care. The role of "mobilizer" would further involve stimulation of other public agencies and voluntary associations to plan and provide needed facilities and services; and to weld those diversified activities into an integrated community attack on the overall problem.

At the national level, we have an interesting example in the experience of the United States with Asian influenza last fall of how effective the mobilization of resources can be. This was a much simpler problem than the complex problems of chronic disease and prolonged disability. The pattern has basic characteristics however which may be adapted to broader problems. The Public Health Service, in the guise of "national" health agency, had direct official channels of information and collaboration with the state health departments, international agencies, the Armed Forces, and the pharmaceutical industry; with the American Medical Association and other professional

organizations, hospital associations, research institutions, and national voluntary health agencies. Added to this were our own not inconsiderable resources, supplemented by an emergency appropriation by the Congress.

Four months from the day we received information on the existence of the epidemic in Asia, a complete national program of vaccine production and distribution by private industry had been consummated. The medical profession, hospitals, industrial medical services, and public clinics were ready to vaccinate large numbers of people according to priorities set up by individual communities. Plans had been completed to carry out a number of important research projects incident to the epidemic occurrence of Asian influenza. The states and communities had developed their plans to be put into effect should the epidemic prove more severe and widespread than we anticipated. Thus, the medical profession, the hospitals, community groups, and the public knew well in advance what would be expected of them in the event the epidemic took on the proportions of a disaster.

The whole effort was voluntary and cooperative, with no one organization, public or private, dominating and exercising control. It was a big job and one well done in joint planning and action by the widest possible variety of national, state, and local organizations.

The development of preventive programs in the field of chronic disease cannot as yet take on such intensive drive and swift accomplishment. Public health agencies, however, are downright stubborn in their devotion to the concept of prevention and even to the idea of combating chronic diseases in the mass. Someday that hope will be realized.

Perhaps its fulfillment is not far distant for at least two major chronic ailments. For example, the cholesterol blood level can now be lowered by severe restriction of fat in the diet or by adding to the diet certain unsaturated fatty acid supplements. These findings have enormous public health implications in the attack on atherosclerosis with its strong pertinence to the problem of coronary disease. Knowledge such as this imposes obligations on public health organizations to use it if the burden of this disease might be lifted thereby. It also demands that we be doubly sure that mass application of such knowledge would benefit the Nation and not do harm. It raises many questions. Would permanent change in the American dietary pattern adversely affect other metabolic processes? What would be the specific effects on atherosclerosis? Would it reverse or slow progression of the disease in persons already affected? Would it prevent atherosclerosis in healthy individuals or merely postpone development of the disease? To obtain answers to these questions, large-scale, long-term studies are essential. But it is stimulating merely to contemplate the possibility of mass prevention of atherosclerosis.

In cancer research there are equally provocative developments. Scientists have been returning with increasing frequency to the role of viruses in the causation of cancer. During the past year, several important advances have been made in this area. At the National Institutes of Health, for example, Drs. Sarah E. Stewart of the National Cancer Institute and Bernice E. Eddy of the Division of Biologics Standards have obtained a viruslike substance from cell suspensions of an experimentally produced parotid-gland tumor grown in tissue culture. This substance has the capacity to produce cancers in many animal tissues. (12) When injected into laboratory animals, it produces multiple primary tumors, including parotid-gland tumors, tumors of the thymus, adrenals, mammary glands, kidneys, and skin. The substance also has the capacity to produce cancers in noninbred strains of mice and in hamsters; that is to say, to cross the so-called species barrier.

We are enormously excited by this important work, since it is the first time that such a wide range of malignant growths has been produced experimentally by a cell-free filtrate having viruslike properties. If this substance is proved to be a virus—if these scientists are able to achieve similar experimental results in material extracted from human tumors—and

if it should be thus established that certain forms of cancer are viral in origin, the whole picture of possible prevention of cancer changes. New vistas of research would be opened in the fields of immunology and epidemiology, possibly leading to new public health methods for the prevention and control of cancer.

Changing Patterns of Hospitals and Medical Facilities

Changes in hospital care are no less striking than those in population, morbidity and mortality, and in the science and practice of medicine and public health. The changing pattern of hospitals and medical facilities is probably more closely linked to socio-economic factors than any other one sector of health service in the United States.

Obviously, the growth and shifts in population have produced needs for hospital construction in the past decade on a scale comparable with the needs for housing, schools, and other basic community facilities. The increase in hospital construction, however, is not the only important change.

The costs of hospital care have risen steeply and continue to rise. The Consumer Price Index shows, for example, that hospital room rates rose by 265 per cent in the 20-year period, 1936–1956, as compared with 73 per cent for general practitioner's fees, and 60 per cent for surgeon's fees. (13) Excluding hospitalization, the price increase was smaller for medical care than for any one of the major groups of commodities and services purchased by the American people. It should be noted, however, that the increase in total medical costs has been greater and more rapid in recent years. In the past decade, they gained by 50 per cent, as compared with 37 per cent for commodities and 18 per cent for other services.

Private expenditures for medical care and voluntary health insurance have increased from $7.3 billion in 1948 to $12 billion in 1956. Significantly, about three-fourths of the total is paid by consumers out-of-pocket, although the proportion met by insurance benefits had increased from 8 to 26 per cent. About half of the expenditure for hospital services is met by insurance benefits. (14)

Various studies in recent years have thrown light on how the costs of hospitalization are being met for persons in the older age groups. The Health Information Foundation reported that in 1952–53, nearly 6 in every 10 persons had hospital insurance, but that only 3 in 10 of those 65 years and over had such insurance. (15) A study by the Public Health Service (16) shows that the proportion of middle aged and older persons in urban families spending $1,000 or more out-of-pocket for medical care in 1950 was half again as high as in the total urban population. Hospital services account for more than 30 per cent of these direct expenditures, and these families paid more for special duty nursing alone than the average urban family paid for all medical care services. Data compiled by the American Medical Association and the Council of State Governments show that 70 per cent of all hospital care received by persons 65 years and over is in governmental institutions, (17) chiefly in state mental hospitals.

There is growing concern about increased medical costs, chiefly with respect to their impact on both private incomes and tax-supported services as a result of catastrophic and long-term illness. A shift is occurring in both public and professional attitudes toward hospitals, as well as toward nursing homes, homes for the aged, and other institutions for the care of patients needing some type of sheltered environment. The demand is insistent and growing for institutional facilities where specialized personnel and equipment can be made available to physicians and their patients with greater efficiency and at lower costs than are possible in a general hospital. Moreover, an increasing number of communities are following Philadelphia's example in the establishment of home care programs as a means of reducing the costs of prolonged hospital care.

State and local governments and voluntary organizations have been slow in their response

to the demand for facilities less costly in construction and operation than the general hospital for care of long-term patients and the aged. As a result, proprietary institutions have sprung up in the past decade in response to the demand by families and public welfare agencies for some place to stow aged and infirm patients. At the time of the national inventory in 1954, four in every five nursing homes and related facilities were owned and operated for profit, in contrast with about one in five of general hospitals in the United States the same year. Half of the proprietary nursing homes had been in operation less than four years.

Although there are many bright spots in the picture, it is generally agreed that facilities and standards of care for patients in nursing homes and homes for the aged are inadequate. The problems involved are largely economic, but the solution of the economic problem depends primarily upon the extent to which the medical profession exerts its influence to bring these institutions into the stream of modern medical and hospital practice.

The Nation is already making a substantial investment in care of the aged in nursing homes. Private and public expenditures for this purpose amount to about $550 million a year. (18) This sum is shared just about 50-50 by tax-funds and private expenditures of patients and their families.

The lack of enough high quality institutions of the nursing home type keeps tens of thousands of patients in general hospitals for prolonged periods beyond the time when they need, or can even benefit from "full-dress" hospital services. The lack also places persistent and exorbitant demands upon many communities for additional general hospital beds. It drives up the costs of hospitalization, and denies good care to hundreds of thousands of individuals, especially persons in the later years of life.

The economic problem of adequate care is likely to be acute for older people. It is true that our system of old age and survivors insurance, combined with private pension plans, assures increasing millions of our citizens some personal income in the later years. For those who are not covered by national or state or private insurance, or whose pensions are too small to purchase care, public assistance programs provide limited financial support. The resources of religious and voluntary organizations supported by the public also are available.

Nevertheless, the situation for the majority of disabled older people is forbidding. Prices are high and rising, but the older person's ability to pay remains static at a considerably lower level than that of the physically or financially independent consumer.

The medical profession knows all too well that prolonged illness and the debilitating processes of aging are intimately associated with poverty. We are indeed vulnerable to a charge of being smug and indifferent in our present prosperity if we fail to face up to this challenge and do all in our power to reduce—in so far as our knowledge, skills, and influence permit—the burden of disabling illness. The removal or amelioration of this factor in dependency would contribute directly to the Nation's social and economic health.

The first National Conference on Nursing Homes and Homes for the Aged sponsored by the Public Health Service in February of this year adopted a series of proposals (19) that will, I believe, guide the development of institutional and community services for the chronically ill and the aging for some years.

Among its many actions, the Conference urged Federal legislation which would encourage both public and private financing for construction and renovating of the homes and would recommend construction standards. Concerning the financing of services, it was recommended that costs of care in such facilities be determined through accepted cost accounting methods as a basis for bringing public assistance payments into line with costs.

Another major recommendation was that model laws and regulations should be developed for adoption by states and communities. These laws should stress protection of the pa-

tient's personal interest, dignity and right of privacy, as well as his medical, social and safety needs, and should be enforced by a single state agency with the aid of an advisory body.

The Conference developed a clear-cut classification of institutions that goes farther than any of us had anticipated. To the traditional residential, personal, and nursing care institutions was added a new classification—the multiple service institution. This would include all types of care encompassed in the others and would emphasize social and groupwork services, auxiliary medical services and rehabilitation.

The Conference emphasized the many opportunities that all professional groups, all teaching institutions, and all community organizations have to enter actively into the development of nursing homes and homes for the aged: The professional schools to increase opportunities for training in care of the chronically ill and aged; community agencies, especially the public health departments, to develop and provide consultative services for nursing homes. A proposal was adopted calling for the licensing of nursing home administrators in line with the licensing of other groups in the health and medical professions. The necessity for continuous medical supervision in all such institutions was emphasized.

As we survey the whole scene of health and medical care in the United States, however, one fact stands out boldly. The hospital holds a key position and plays a paramount role in any effort to extend and improve the care of the chronically ill and the aging, and to reduce the costs of care. Our hospitals have grown to the point where they comprise a big industry. They account for $6 billion a year in the Nation's gross product or expenditure. They employ about 1,400,000 people, as compared with some 650,000 in the steel industry, and 600,000 to 800,000 in the automobile industry.

A business of such size and importance could not long survive in the modern age without continuous and substantial research and development as a basis for operations. Hospitals naturally depend upon scientific advances in related fields to improve the care of patients, but an annual expenditure of $3 million from all sources for specific hospital administrative research would be a generous estimate indeed. This may be compared with the national investment of $360 million in medical and biological research. The Public Health Service's research grants program contributes less than $1,200,000 directly to hospital research, although some of our own studies conducted in cooperation with institutions, and some grants through the National Institutes of Health are related to hospital research.

Despite the many obstacles I have mentioned, new patterns in the organization and administration of hospitals are beginning to emerge. Patterns of regional coordination have multiplied and are being extended. The work of the Hospital Council of Philadelphia, for example, is one of the early efforts in this direction.

Some general hospitals scattered across the country have experimented with "self-service" or minimal care units for patients not requiring the maximum of nursing and related services. A few have constructed annexes for convalescent care or facilities of the nursing home variety for long-term patients. Some mental hospitals have developed outpatient departments; some are experimenting with "half-way houses", foster home programs and the like. In many general hospitals, departments of physical medicine and rehabilitation have been strengthened. The concept of the comprehensive rehabilitation facility under hospital auspices is gaining wider acceptance.

At present, interest is growing in a more fundamental type of reorganization of the acute general hospital. Since so little knowledge has been accumulated, the term "progressive medical care" has been attached to the emerging concept.

In most general hospitals, services are based on a single, rigid concept of the patient as a person flat on his back in bed throughout his hospitalization. This means that the hospital's readiness-to-serve costs have to be estimated

at a maximum, so as to extend, presumably, the same type of care to patients who actually present a very wide range of needs. As a result, the service may be too elaborate for some patients; those who are hospitalized for a diagnostic work-up for example, or those who no longer need or can benefit by complete bedside service but still require hospitalization. As practicing physicians know, this often spreads the service so thin that acutely ill patients do not recieve sufficient intensive care.

The concept of "progressive medical care" in hospital organization revolves around the medical and nursing needs of the individual patient. It envisions an intensive care unit where acutely ill patients in groups of 4 to 6 can be given constant nursing attendance and where the life saving devices are always at hand. When they no longer require such intensive service, they would progress to an intermediate care area. Thence, if further hospitalization is required, they would go to a self-care unit where they can have their clothes, get about on their own and go to the cafeteria for meals—a better environment for this stage of recovery. Adjoining the hospital would be a nursing home type of facility for long-term patients. All these units would be linked to a home care program and ambulatory services. Rehabilitation services would be available for each "progressive" area. Thus, the emerging pattern of hospital and medical facilities would link together five types of service for the patient and his physician: Intensive care, intermediate care, self care, long-term care, home and ambulatory care.

The Public Health Service is conducting a study of some 7,000 hospitals to determine the extent to which one or more of the five types of care have been incorporated into hospital practice. We expect to complete this study and make the findings available some time next fall.

We are also conducting a research project in cooperation with the Manchester Memorial Hospital at Manchester, Connecticut, where the three intramural types of progressive care —intensive, intermediate, and self care—were initiated about a year ago after a careful analysis of the situation under the traditional type of hospital organization. This hospital is serving as a pilot plant, so to speak, as when in industry the problems related to a new industrial process are studied intensively and methods for their solution evolved.

Another research area that holds much promise is in the adaptation of modern industrial and business administrative methods to the hospital setting. Some hospitals have been reluctant to adopt administrative principles and procedures that have been so successful in other organizations. Yet they are constantly faced with demands to improve services and deliver them at lower costs. There are several projects of this sort underway with Public Health Service grants.

Health and hospital authorities all over the country are following with keen interest the Philadelphia Hospital Council's study of the growth and development of private physician's offices in hospitals. I am pleased that this study is being supported by a Public Health Service research grant. Dr. Rufus Rorem and your Council will make a real contribution to hospital progress through a thorough analysis of this little-understood pattern of hospital and medical care organization.

As you are aware, the American College of Physicians is also conducting a major study in the field of hospital research. The aim here is to develop standards for departments of internal medicine. The Joint Commission on Accreditation heretofore has had to depend largely on the adaptation of standards developed for departments of surgery. Surely it is desirable that internal medicine develop specific standards in its own field.

Conclusion

I have tried to present to you some of the momentous changes in our national life which are proceeding at a rapid pace and which affect especially the practice of medicine and public health. In our professions, it often happens that great changes, however striking and far-reaching in their effects, catch us unaware.

If we are to bear our share of the responsibility in influencing change in a forward direction—a direction toward medical progress and toward better health for the American people, we must be prepared to become involved in the larger life of our communities and our country. Surely no physician in the modern period did more to teach his colleagues that lesson than did James M. Anders. He was involved in every aspect of his community's life and he extended that personal involvement to the national scene in his work with many professional and civic organizations. He was prepared for change, welcomed and stimulated change.

I can find no more appropriate words with which to close than the tribute paid to Dr. Anders thirty years ago by his colleague, Dr. E. J. G. Beardsley, then Professor of Clinical Medicine at Jefferson Medical College.

"To a great many physicians any change from their usual mode of thought and method of practice is difficult if not impossible. Dr. Anders stands for progress in every field and change has no terror for him if the results will justify the changes." (20)

May all of us in medicine and public health hold to that ideal.

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Biological Factors in the Radiation Problem Relating to Society[1]

By CHARLES L. DUNHAM, M.D.[2]

CARLETON COON in his *Story of Man* has said, "Until he found fire, man was nothing but an animal with tools instead of claws and fangs." For thousands of years with the aid of fire to keep him warm, to smelt metal, to drive engines and machines, to hasten chemical reactions and to drive rockets, man has learned to exploit the reaction of combustion almost to the ultimate. Meanwhile from time to time he has employed this power in every way that he could think of in making war. Yet he has survived, regardless of how careless he has been. Atomic energy is made of different stuff. We are going to have to be more respectful of it than we have been of fire and high explosives. It has inherent in it a greater potential for good, but at the same time an equivalently greater potential for harm. Like the other great technical advances, it is bound to exact some price of the society which makes use of it.

By 1902, a bare seven years after the discovery of x-rays by Roentgen the cause and effect relationship between exposure to x-rays or to the emanations of radium and skin cancer was clearly recognized. It has been known since the mid-twenties that radiation of germ cells can result in gene mutation, and radiation exposure during embryonic life can result in developmental abnormalities. Only a few years later, radiation-induced leukemia in mice was observed.

By 1940 the hazards of ionizing radiation were already understood in a general way. From 1929 to 1940 what is now named the National Committee on Radiation Protection and Measurement had been cooperating with the International Commission on Radiological Protection in developing recommendations concerning the maximum permissible exposure of the relatively few adult workers using x-ray machines, radium and later other sources of ionizing radiation. The recommendations were based on scientific facts, obtained both experimentally and by observation of injury incurred by pioneers in radiology and radiological physics and by the workers in the luminous dial industry. Since then as more and more data on the effects of radiation accumulated, these recommendations have been revised. The Atomic Energy Commission, in all its operations, has endeavored to follow these recommendations.

Public concern, and concern by scientists other than radiobiologists, with the effects of ionizing radiation on human beings became widespread as a result of the unfortunate accidental exposure of the Rongelapese to fallout in the spring of 1954 from atomic weapons testing in the Pacific. In June 1956, as a result of the report of the National Academy of Sciences study on the biologic effects of atomic radiation, this concern spread from the hazards associated with radioactive fallout to include the hazards inherent in the medical uses of x-rays and in the coming age of nuclear power production.

One of the great difficulties in discussing this problem at the present time is that of achieving objectivity. Inherent in the present discussions of the effects of radiation is the matter of whether or not the United States should attempt to build optimal capability in the delivery of atomic weapons in the event of war and even as a deterrent to war. The subject has become intimately involved in many people's emotions. During the 1956 Presi-

[1] Presented before the Section on Public Health, Preventive and Industrial Medicine, College of Physicians of Philadelphia, 18 March 1958.

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dential election campaign it became a political issue.

Another difficulty is one of achieving a proper perspective in dealing with the manner in which the delayed effects which might be produced by radiation would appear. The fact that any delayed effects are by definition ones which do not become apparent at once is baffling to the majority of people, and the fact that they could be produced by something as intangible as radioactive atoms lends the subject a weirdness that is hard to dispell.

We have a situation quite the reverse to what has pertained in the past with respect to the great epidemic diseases. Smallpox, cholera, and typhoid fever epidemics were very real and terrible events which decimated whole populations in devastating fashion. The problem was to seek the cause and eliminate it by sanitation or by immunizing the population to the specific causative organism. With radiation hazards to society as a whole it is otherwise. There are no formidable pressing measurable effects for which to seek a cause. We can with varying degrees of confidence predict effects from the present rate of radiation exposure to the population that can never be measured or clearly identified with the specific cause. Yet each predicted effect is described in the form of some well-known tragic event—a deformed or weakened child—leukemia, bone cancer or premature death.

The problem is to find some means of comparing the hazards inherent in fallout and in the medical and industrial uses of atomic energy with some more familiary man-made hazard which is presently tolerated for one reason or another. There is a natural tendency to reject comparison with the hazards from such a familiar thing as fire. Fire exacts 10,000 lives a year in this country and at the present rate 300,000 per generation. It scars and maims many thousands more. There is the same tendency to reject comparison with the automobile—a very real symbol of modern civilization and the cause each year in the United States alone of some 40,000 deaths and a like number of maimed—at the present rate more than a million killed per generation. Similarly, people reject comparison with the accidental deaths and injuries which are very substantial and a sine quo non for our defense effort. I am sure that you will agree with me that these figures are needlessly high, and must and can be reduced, just as injuries from radiation must be kept to a minimum. I suspect one of the reasons for the complacency about our present accident rate is the commonplace nature of the incidents which lead to death and injury. They are recorded in our newspapers daily. A hazard more comparable to that of radiation—smog or air pollution—cannot be used for comparison simply because we have no comparable body of knowledge upon which to base an estimate of the possible deleterious effects on our citizens. For radiation and especially radiation from fallout constitutes the only contemporary man-made general environmental hazard about which we have sufficient information to define it at all.

Nevertheless, just because it can be defined there is a greater obligation to keep radiation hazards minimal.

The present rate of exposure from medical and dental x-rays (4R in 30 years) is of about the same magnitude as the exposure from natural sources of radiation. The present levels of radiation exposure from weapons testing fallout (0.13 R in 30 years) and future levels at any realistic rate of weapons testing, whether by one nation or by many, are even lower, they are a fraction of the natural radiation exposure. In fact, these levels are well below the levels of radiation which have been employed in experimental work in order to demonstrate detectable pathologic or genetic changes.

Genetic Effects

In the field of genetics there are two principal hazards with which we are concerned when we study the effects of ionizing radiation as a mutagenic agent. First, there is the possible risk to the human race as a whole. There is undoubtedly some amount of radiation which, if the entire race were subject to it, would re-

sult in a mutation rate which would lead eventually to degradation of the species. On the other hand, the maximum tolerable mutation rate for humans, tolerable in the sense of survival of the race, is not known. It has been generally agreed by the National Academy of Sciences Committee on the Effects of Atomic Radiation that increases in the mutation rate which would result from an additional average exposure, above natural radioactivity, of 10 R to the gonads per generation would be tolerable but not harmless.

The other hazard is to some individuals, the personal tragedies associated with the birth and life of a defective child. The NAS Committee has estimated that in the normal course of events in the next 30 years 100,000,000 children will be born in this country and that there will be among them some 2,000,000 with tangible genetic defects. If 40 R is taken as the radiation dose per generation necessary to double the present "spontaneous" mutation rate, the 10R dose per generation mentioned in the NAS Report would add in the United States alone 50,000 tangible defects in the first generation and eventually after 20 or 30 generations about 500,000 per generation, i.e., about 16,000 per year.

A dose of 0.13 R to the gonads per U. S. generation is estimated to be incurred from the present rate of weapons testing. This is estimated to produce in the first generation an additional 650 persons with tangible genetic defects, and if this rate of exposure continued there would eventually after some 20 to 30 generations be 6,500 per generation. There would be in addition about 5,000 embryonic and neonatal deaths, stillbirths and childhood deaths in the first generation with eventually about 80,000 per generation. There would also be a larger but unknown number of minor intangible defects. Were the dose received by the world as a whole the same (actually it is lower), you would have to multiply all the figures by about 20. In absolute numbers they are large. On the other hand, when one compares them with the 2,000,000 tangible genetic defects which are now occurring in each generation and millions of embryonic, neonatal deaths, stillbirths and childhood deaths from genetic causes, they are a small fractional increase. The effects of medical x-rays could be said to be adding eventually about 1/10th to the present U. S. total, while fallout at the present rate of testing would add an increment of 1/300th.

Life Span

To the best of our knowledge, except for high level radiation to vital organs, the life shortening effects of ionizing radiation are the result of total body exposure or they may manifest themselves in succeeding generations as a result of genetic damage. There is considerable experimental data in small mammals on the effects of fairly large single event whole body exposure, i.e., one to two hundred R and more given either once or repeated. There is considerably less information at smaller dose increments. In general, it can be said that with large increments, 100 R and more, there is a curtailment of life expectancy from the time of exposure by approximately 6% per 100 R. Thus a single dose of 200 R would be expected to reduce the average individual's life expectancy from that point on by roughly 12.5%. With smaller increments, a few R to upwards of 100 R, the effect in experimental animals is less marked. One explanation for this is the possibility of a partially effective reparative process. The curtailment of life expectancy is in these circumstances a little less than 1% per 100 R. If this holds for human beings an average individual who had accumulated at the age of 40 years approximately 100 R in increments of several roentgens at a time and who would normally be expected to live another 30 years would lose 3–4 months of his life span.

There is no definitive information at low dose rates, i.e., 0.1 R per day or 0.3 R per week and less which is in the range of the permissible exposure levels as recommended by the International Commission on Radiological Protection. A few experiments have been done in mice and rats. In each instance the average

life span of the irradiated group was slightly higher than that of the control. It appears, however, that the sparing effect is during middle life, and perhaps, chronic low level exposure has some sort of non-specific effect by permitting survival of experimental animals in the presence of certain ectoparasites. The longer-lived animals in the irradiated group did not live any longer than the longer-lived animals in the control group. In any event, a dose rate of 0.13 R per 30 year period would reduce the average life span by less than a day. While the dose rate of 4 R per thirty year period from diagnostic X-rays might curtail the average life span by at most two or three weeks.

Leukemia

The present leukemia rate in the United States is approximately 11,400 cases per year. It is an established fact in many experiments done on animals that large doses of radiation do induce leukemia. In some experiments, although the total number of cases was not increased, the onset was greatly accelerated by the radiation exposure. For doses of less than 100 R in humans and in statistically significant numbers of experimental animals, there are no definitive data. The available experimental data from fairly extensive studies at higher levels of radiation suggest that depending on the type of leukemia the induction curve may be either sigmoidal or linear. It is not known whether or not there is a threshold for leukemia induction by radiation. While it has been generally accepted among students of leukemia that there is some accumulated dose of radiation, perhaps in the vicinity of 50 R below which leukemia is not induced, Dr. E. B. Lewis of California Institute of Technology and Dr. Hardin Jones of the University of California at Berkeley have proposed the hypothesis that leukemia induction from ionizing radiation is a linear function of dose regardless of dose rate and have suggested that for each MR average exposure per year to the entire population of the United States, there would be eventually be an additional 10 cases of leukemia per year, i.e., about 40 cases per year from fallout. Using this same reasoning there would be roughly 1200 cases per year as a result of diagnostic medical X-rays.

It has also been postulated that bone-seeking radioactive nucleids such as radiostrontium might be leukemogenic. If Lewis' hypothesis is correct, that leukemia induction is linear with dose to the bone marrow, and were all the bone marrow to receive this dose which it does not, such a body burden for all people in the United States could mean an additional 5–10% increase in leukemia (500–1000 cases each year). There is however a considerable body of experimental data indicating that with large single doses leukemia does not result if a fair fraction of the hematopoietic system is shielded from total body radiation. With a certain type of mouse lymphoma, even shielding one extremity of the animal will vitiate the leukemogenic effect of a large single exposure to radiation.

It appears then that if leukemia in general or even one type of leukemia can be the result simply of a radiation-induced somatic mutation untempered by homeostatic factors, fallout at the present rate of weapons testing could, on the basis of certain assumptions as to the number of cases of leukemia due to background radiation, result in this country in some 30–40 additional cases per year or about 1000 per generation. The same assumptions lead to a figure of 1200 cases per year as the result of medical X-rays (36,000 per generation). If small amounts of strontium-90 relatively uniformly distributed in bone can also produce leukemia in the manner postulated by Lewis, fallout from continued weapons testing at the present rate could eventually lead to 35 to 250 additional cases per year (1000 to 7000 per generation). To complete the story, one must keep in mind that cocarcinogenic factors and additive factors may in certain susceptible individuals prepare the way for a small dose of radiation to trigger a case of leukemia.

Bone Cancer

The present incidence of bone sarcoma in this country is about 2000 cases annually. It is apparent from the observations of radiotherapists that a dose of something more than 1000 R given locally to the bone is required to induce cancer, and cancer induction by doses of less than 2000 R is a very rare occurrence. As to the induction of cancer by chronic irradiation from bone-seeking radionucleids, we have a considerable body of data in human beings. Briefly, it can be stated that no case has come to our attention of bone cancer in an individual exposed to radium in adult life who had left in him at the time of observation (usually 20–30 years after the material was ingested) less than 0.4 microcuries of radium. The National Committee on Radiation Protection and the International Commission on Radiological Protection have taken 0.1 microcurie as the permissible radium burden for adult workers. The corresponding figure for strontium-90 is 1. microcurie. One tenth of that or 100 $\mu\mu$c of strontium-90/gm calcium, the presently considered permissible body burden for the population as a whole, would give about 0.28 rad per year or 20 rad in 70 years, i.e., approximately three times the exposure to bone from naturally occurring radioactivity.

Experimental work in mice and dogs at such low body burden levels is incomplete, but at somewhat higher levels of strontium-90 in mice the curve for bone tumor production is a steep sigmoidal one. In other words, very few if any bone sarcomata will result.

I have attempted to review for you in an objective way the best information now available on the possible cost to society of the privilege of making use of atomic energy. The estimate for the genetic cost in terms of point mutations is based on a wealth of scientific data and while not absolutely proven the burden of proof should lie with those who question it. The basis for the estimate of the upper limit for the leukemia cost is still in the realm of hypothesis though it must be taken account of in computing a possible maximum cost of nuclear energy. Estimates of the cost in terms of shortening of the average life span but exclusive of genetic effects as they may affect the life span of future generations are based on good data from experiments in animals exposed to dose increments of several roentgens. Whether there is any effect on life span at very low dose rates is not known.

The cost estimates discussed, even including the estimate for the ultimate genetic cost which is at least an order of magnitude greater than the highest cost estimates for leukemia, are well below our present day experience with accidental deaths which is admittedly higher than need be. All of these estimates assume that there will be no further advances in the biological sciences with respect to the prevention and treatment of leukemia and cancer in general, and in our ability to counteract or protect against the mutagenic effect of ionizing radiations. In other words, it is possible to live with nuclear energy and to exploit it fully at what society may accept as a reasonable cost.

Nuclear energy has presented civilization with perhaps its greatest challenge since the discovery of fire. We have within our grasp an ability undreamed of a century ago to control our environment. We have a key to almost unlimited power. What we do with all this depends on us. If we are careless with nuclear energy we can injure ourselves seriously. If we grossly abuse it we could probably destroy our present civilization.

In our enthusiasm for the fruits of science and in our newly recognized need to keep in the forefront of world science as a means to survival we, and here I mean not just the United States but the entire civilized world, must not be blinded to the fact that unless we very rapidly achieve a much greater maturity in the political and social sciences we shall find that we have built for ourselves a fool's paradise. Whatever we do to our educational system to stimulate science, it should not be at the cost of the humanities and the social sciences; for if we do it that way we shall certainly pay for it very dearly.

Neonatal Mortality in Philadelphia[1]

By NORMAN KENDALL, M.D.[2]

THE DECREASE in neonatal mortality in Philadelphia over the past few decades has failed to keep pace with the decrease in maternal and infant mortality. This continued high neonatal mortality rate is in part needless and presents a challenge to the medical profession. A program designed to lower the neonatal mortality rate can only be formulated when the facts and data are known. The following is a partial analysis of the neonatal deaths in Philadelphia during 1955.

In Table I are shown the total number of births and the neonatal mortality rates per thousand live births in Philadelphia from 1950 to 1955. The total number of births has been steadily increasing. The lowest neonatal mortality rate during these years, however, was in 1951, when 20.9 infants per thousand live births died during the first twenty-eight days of life. Subsequently, the neonatal mortality rate has gradually risen to 23.1 in 1955. The premature birth rate in Philadelphia for the years of 1954 and 1955, as shown in Table II, is above the national average of six to seven per cent; the premature birth rate for the non-white population is twice that of the white population.

Neonatal deaths in Philadelphia during 1955 are analyzed according to birth weight and race in Tables III, IV and V. Seventy-five per cent of these deaths occurred in premature infants (birth weight less than 2500 grams or five and one-half pounds). Almost one-third (thirty-one per cent of all deaths) occurred in infants who weighed less than 1000 grams (two pounds and three ounces) at birth. Although the neonatal mortality rates for both full term and premature infants were comparable in the white and non-white population, the high premature rate of the non-white population was responsible for a neonatal mortality rate of 33.4 per thousand live births in this group, as contrasted with 19.2 per thousand live births in the white population. Although only 8.4 per cent of the premature infants weighed less than 1000 grams at birth, this group was responsible for 41.5 per cent of the deaths of the premature infants. The mortality rate for infants weighing less than 1000 grams at birth was approximately 95 per cent; for the group of infants weighing between 1001 to 1500 grams, it was 64.3 per cent. This latter figure is excessively high and may reflect inadequate care. Infants weighing between 1501 and 2000 grams at birth have a much better chance of survival, the mortality rate having fallen to 17.8 per cent.

The neonatal deaths according to age at death and weight at birth are shown in Table VI. Approximately forty per cent of the deaths of full term infants occurred in the first 24 hours of life, whereas fifty-eight per cent of the deaths of the prematurely born infants occurred during this period. Of the full term infants, 25.2 per cent of the deaths occurred between the second and fourth week of life. This is certainly an excessive loss, and warrants further investigation.

A comparison of the neonatal mortality rates for New York City, Chicago, Philadelphia, the State of Pennsylvania and the United States (Table VII) for the years 1950 to 1955 reveals that the rates for Philadelphia are consistently higher than are those of the other cities, of Pennsylvania, or of the country as a whole.

In Table VIII are shown the neonatal mortality rates, the percentage of premature births and the percentage of illegitimate

[1] Presented before the Section on Public Health, Preventive and Industrial Medicine of the College of Physicians of Philadelphia, 21 January 1958.

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TABLE I

NUMBER OF BIRTHS, DEATHS AND NEONATAL MORTALITY RATES FOR PHILADELPHIA, 1950–1955

Year	Births	Deaths	Rate/1000 Live Births
1950	47,516	1,058	22.3
1951	50,318	1,054	20.9
1952	51,661	1,113	21.5
1953	51,706	1,162	22.5
1954	53,716	1,209	22.5
1955	52,368	1,209	23.1

TABLE II

PERCENTAGE PREMATURES ACCORDING TO RACE, PHILADELPHIA, 1954 AND 1955

Year	Total	White	Non-White
1954	8.7	6.9	13.8
1955	9.3	7.0	14.5

TABLE III

NEONATAL DEATHS BY BIRTH WEIGHT, PHILADELPHIA, 1955

Weight, Gm.	Number	Per Cent
2501+	286	23.6
2500−	907	75.0
Unknown	16	1.4
Totals	1209	100.0
2001–2500	99	8.1
1501–2000	156	12.9
1001–1500	276	22.8
1000	376	31.1
Totals	907	74.9

TABLE IV

NEONATAL MORTALITY BY BIRTH WEIGHT AND RACE/1000 LIVE BIRTHS, PHILADELPHIA, 1955

	All Races	White	Non-White
Total	23.1	19.2	33.4
2501 gm−	191.9	190.2	194.1
2500 gm+	6.0	6.0	5.9

TABLE V

NUMBER AND PERCENT OF PREMATURE BIRTHS AND NEONATAL DEATHS BY WEIGHT, PHILADELPHIA, 1955

Weight, Gm.	No. Born	Per Cent	No. Died	Per Cent	Per Cent of Premature Neonatal Deaths
2500−	4726	100	907	19.2	100
1000−	396	8.4	376	94.9	41.5
1001–1500	429	9.1	276	64.3	30.4
1501–2000	875	18.5	156	17.8	17.2
2001–2500	3026	64.0	99	3.3	10.9

TABLE VI

NEONATAL DEATHS BY AGE AT DEATH AND WEIGHT AT BIRTH, PHILADELPHIA, 1955

Age at Death	Total	2501 gm+	2500 gm−
Under 2 hours	15.6	13.6	15.6
2–23 hours	38.9	27.6	42.8
1–6 days	32.4	33.6	32.2
7–27 days	12.9	25.2	9.2

TABLE VII

COMPARATIVE NEONATAL MORTALITY RATES/1000 LIVE BIRTHS, 1950 TO 1955

	New York	Chicago	Phila.	U.S.A.	Penna.
1950	19.0	19.3	22.3	20.5	20.7
1951	18.4	20.1	20.9	20.0	19.1
1952	18.3	18.8	21.5	19.8	19.2
1953	18.2	19.4	22.5	19.6	19.4
1954	18.0	19.2	22.5	19.1	19.0
1955	20.0	18.8	23.1	19.1	19.2

TABLE VIII

NEONATAL DATA, PHILADELPHIA, BY RESIDENCE, 1955

Health District	Neonatal Death Rate	Percent of Premature Births	Percent of Illegitimate Births
1	34.2	11.6	22.2
2	24.8	8.2	5.5
3	24.0	6.5	9.5
4	37.5	10.6	12.8
5	33.6	12.9	17.6
6	30.7	11.6	15.4
7	19.0	6.6	1.0
8	18.9	6.9	2.5
9	19.3	6.7	2.2
10	17.5	5.2	0.3
	26.0	8.7	8.5

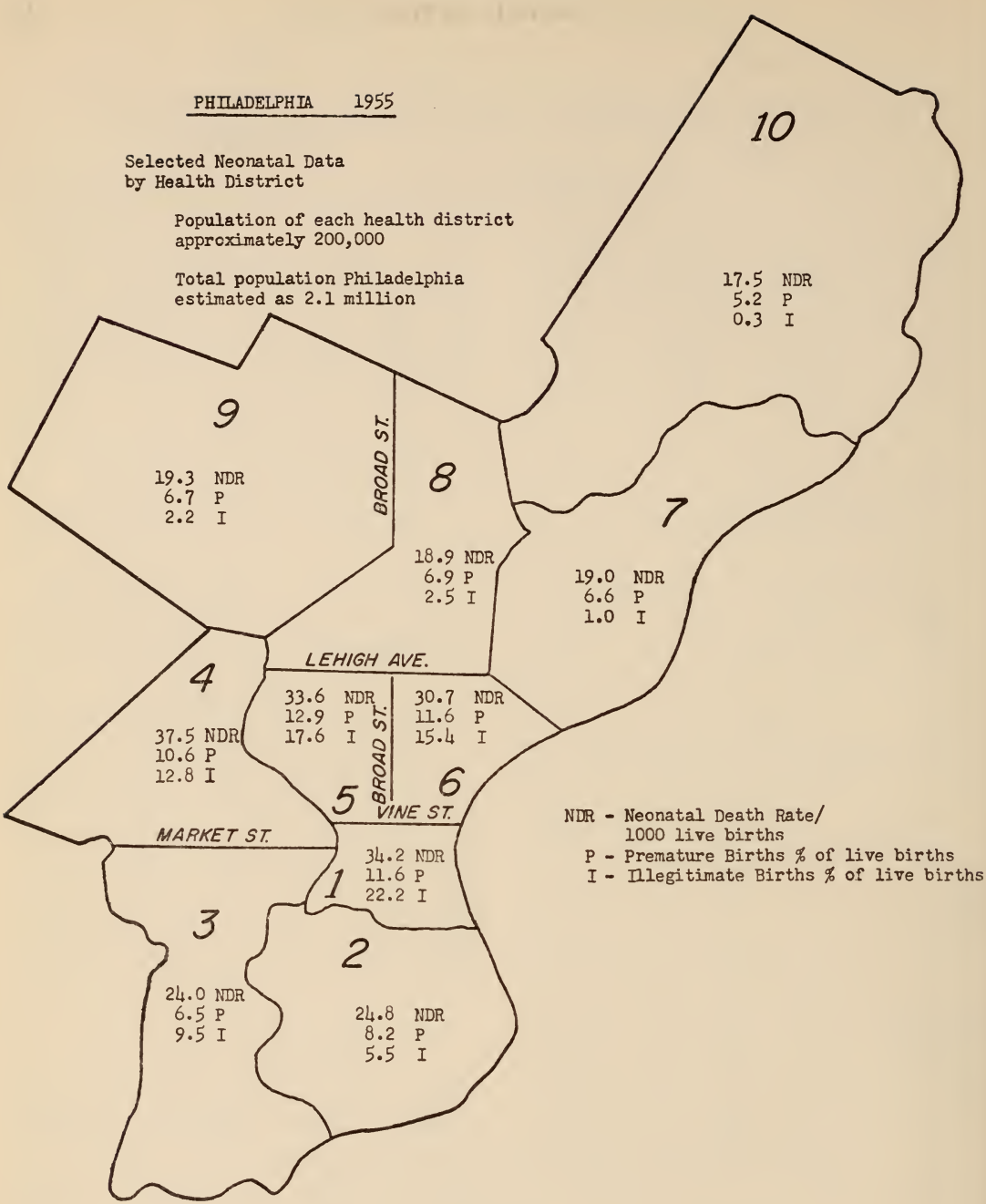

FIG. 1

births according to health districts in the City of Philadelphia. Figure 1 illustrates the geographical distribution of these health districts in the city. It is obvious that the premature rates and neonatal mortality rates are increased in those areas in which there is a high rate of illegitimacy. In Philadelphia Health Districts 7 to 10, the neonatal mortality rate is less than 20 per thousand live births; the rates for prematurity and illegitimacy are

correspondingly lower here than in the other health districts. There appears to be a distinct relationship between these three factors.

A realistic approach to decreasing the neonatal mortality rate in Philadelphia must be directed at illegitimacy and the premature infant. In 1955 seventy-five per cent of the neonatal deaths occurred among the premature infants and approximately thirty-two per cent of the neonatal deaths were among infants who weighed less than 1000 grams at birth. The chances for survival of a premature infant increase many fold if the period of gestation is prolonged from 28 to 32 weeks or longer. It remains for the obstetrician to attempt to prevent premature labor and/or to prolong the period of gestation so that the chances for survival of the infant are enhanced.

There is a large area for improvement in the care of the premature infant. Improved nursing care and better facilities should be provided. A training center for the education of nurses in the more advanced and modern methods of the care of premature infants should be established. Many of the smaller hospitals, owing to a lack of nursing and medical (intern or resident) care, cannot now adequately care for the small premature infant. For this reason, several premature reception centers should be established within the city. Premature infants weighing less than four pounds could be transferred at 48 hours of age to these centers, where adequate medical and nursing care would be available on a twenty-four hour basis. The lower premature mortality rates in Chicago and New York as compared with Philadelphia may be in part the result of such programs.

The revision of *Regulations for Maternity and Nursing Practices in Hospitals* by the Philadelphia Department of Health is long overdue. When these revised regulations have been adopted, a program for inspection of hospital nurseries can be formulated. The inspection team should serve in a consultative rather than in a punitive capacity, and should be designed primarily to assist the hospitals in dealing with broad problems concerning the care of newborn infants. For example, the training and experience of such public health officials as would be available in such a team, would be of great assistance to hospitals today in establishing the epidemiologic factors of staphylococcal infections which are now all too frequently encountered in some of the nurseries in the city.

The high rate of prematurity among the non-white population in Philadelphia is probably responsible for the high neonatal mortality rate. Although many Negro infants tend to be smaller at birth than white infants of comparable gestational age, the high rate of prematurity among the non-white population is undoubtedly closely related to social and economic factors. A broad program of health education must be presented to this segment of the population, since the medical facilities which are available are not now being utilized to the fullest extent. As an illustration, a large segment of this socio-economic group do not have any prenatal care, and the first time that a woman sees a physician during her pregnancy is when she presents herself at the hospital in labor. The lack of prenatal care is a major contributory factor in the complications of pregnancy, in the occurrence of prematurity and in the increase in neonatal mortality. The social agencies and the churches in the city must be consulted and methods devised whereby the importance of prenatal care and of health education can be adequately presented to this group at the earliest opportunity.

Women Physicians and the Medical Societies[1]

By CATHARINE MACFARLANE, M.D.[2]

PRESIDENT RHOADS, Dean Fay, Fellows of the College of Physicians of Philadelphia, and Guests:

On contemplating the important subject I have been invited to present, I am reminded of the young lady from Merion who was being presented at the Court of St. James. On being asked "What part of the United States do you come from?", she replied "from the Main Line."

Since you all know that I come from Philadelphia and the Woman's Medical College of Pennsylvania, you will not expect me to cover the whole field.

Many years ago, far back in the Middle Ages, a group of learned gentlemen after serious consideration decided that women had souls. As century succeeded century, similar groups of learned gentlemen concluded that women had minds. One of the last strongholds of scepticism concerning the mental and spiritual qualifications of Adam's missing rib was furnished by the medical societies. Perhaps the members regarded them as private clubs where, as a leading member of the Philadelphia bar once told me, "Women cease trouble and the men can have a rest."

In 1850, one hundred and eight years ago, in a three roomed house at the rear of a red brick mansion at Third and Arch Streets in this city, the Female Medical College of Pennsylvania was established. It was the only College in the world for the education of women in medicine. The leading spirits in the venture were Dr. Bartholomew Fussell, a Quaker physician, and William J. Mullen, a successful business man and champion of unpopular causes.

Year after year, as young women graduated from this College, they looked forward to joining the Philadelphia County Medical Society, the Medical Society of the State of Pennsylvania, the American Medical Association, just as their brothers or cousins or boy friends graduating from Penn or Jefferson did. To the young women, this seemed a natural and laudable ambition. Not so to the already entrenched members of these societies.

Opportunity came first in Montgomery County where the Montgomery County Medical Society was founded in 1847. In the transactions of the Society we read:

> "Norristown, May 1870. Doctress Anna Lukens was elected a member. This is the first female physician ever elected in a county society in Pennsylvania, or perhaps in the United States—perhaps in the world. She was a pupil of Dr. Hiram Corson and is a graduate of the Woman's Medical College, Philadelphia."

After these proud words the record continues:

> "Dr. David Schrack read a lecture on "The Past and the Future"—a pleasant and instructive address."

This seemed a very suitable topic to follow what, in today's language, might be considered the launching of a medical "Sputnik." The record does not tell us if Doctress Anna Lukens was young or old, how she looked, how she dressed—nothing. Nevertheless she made history eighty-eight years ago. Remember the name Hiram Corson. We shall meet him again. He too was a Quaker, a champion of unpopular causes, one of the best friends the women physicians ever had. As Theodore Roosevelt once said "Success in life depends upon ability to make friends."

The Philadelphia County Medical Society was organized in 1848. In 1853, when the women doctors began knocking at its doors,

[1] Read before the Section on Medical History, College of Physicians of Philadelphia, 21 January 1958.

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it had 220 members. Most of these were opposed to women physicians. In 1868, the Society took firm action, deciding that its members should not consult with "females" and that, if any member accepted a chair on the Faculty of a Woman's Medical College, his membership would be forfeited.

However, "Time marches on." Thirteen years later, in October, 1881, the following resolution was passed:

"RESOLVED that female medical practitioners in good standing in the profession are eligible to membership in this society under the same laws and regulations now governing the admission of men."

Actually it was seven years later, in 1888, that a woman was admitted to membership. She was Dr. Mary Willets, a graduate of the Woman's Medical College of Pennsylvania. As a former President of the Society once told me: "In 1888 the first black man and the first white woman were admitted to membership."

This goal achieved, the women physicians of Philadelphia County became eligible to membership in the Medical Society of the State of Pennsylvania and the American Medical Association.

In 1958, the Society has 3,600 members, of whom 243 are women. The women attend meetings with average regularity. They take part in business and scientific programs, serve on committees and as delegates.

The Medical Society of the State of Pennsylvania was also founded in 1848. In a prefatory note to the centennial anniversary volume, the editor, Dr. Howard K. Petry, points with pride to—among other things—the Female Medical College of Pennsylvania established in 1850, the first Woman's Medical College in the world, and to Clara Swain, who graduated from this College in 1869, and became the first woman medical missionary in history.

However, succeeding pages of the anniversary volume recording the first 48 years of the Society show no such enthusiasm. In fact, the subject of the female physician seemed to be definitely annoying. In 1859, 1860, 1865, 1867, 1871, the *Transactions* record that Dr. Hiram Corson or Dr. Washington Atlee or Dr. Trail Green sought to obtain this objectionable person's admission to membership in the Society but without success. In 1865, in an effort to get rid of her, the Society voted to refer the subject of the female physicians and their membership to the county societies.

All this has changed. In 1958, women take an active part in business and scientific sessions, sit in the House of Delegates, serve as officers, as secretaries and chairmen of sections and panels, serve on committees and commissions. Last year I was honored by being appointed Chairman of the Commission on Cancer.

The first woman to be admitted to the American Medical Association was Sarah Hackett Stevenson, a graduate of the Woman's Medical College of Chicago. She was sent as a delegate to a meeting of the Association held in Philadelphia in 1876. The Philadelphia women physicians did not become eligible for membership until 1881.

In 1958 there are 170,000 members of the American Medical Association; of these about 6% are women. These women take an active part in the scientific programs of the Association, present exhibits, preside at section meetings, serve in the House of Delegates.

The Obstetrical Society of Philadelphia was organized in 1868, largely through the efforts of Dr. Albert H. Smith, a prominent obstetrician and gynecologist, and a Quaker. Dr. Smith was a loyal supporter of the Woman's Medical College, and consultant to the staff of the Woman's Hospital affiliated with the College. From time to time at meetings of the Society he read papers prepared by women physicians or showed interesting specimens from the Woman's Hospital. At a meeting of the Society on October 2, 1879, Dr. Smith read the following resolution and requested that it be placed with the notice of the next stated meeting for action:

"MOVED that when any physician in

regular standing shall announce for presentation to a meeting of the Society through a member, a specimen for exhibition or paper to be read connected with the object of the Society, said Physician shall be considered a guest of the Society at such meeting and may be invited by the President to enter into discussion to which such presentation may give rise."

The request of Dr. Smith was agreed to and at the November meeting his resolution was offered and seconded by Dr. Ellwood Wilson. Dr. Ingham stated:

"There being nothing to prevent the presence of women as visitors and only the impossibility of obtaining the necessary four-fifths votes prevents them from being members, as an expression of opinion I would take pleasure in voting for it. Women had already obtained admission to a number of medical societies and were valued members. Science should take no recognition of sex or color."

Dr. William Goodell, a former President of the Society, said:

"My education, my associations and feelings would make it painful to me to meet women in this Society."

Nine members voted in favor of Dr. Smith's resolution, eighteen voted against it.

For the next thirteen years, the minutes of the Society make no mention of women physicians as guests, members or otherwise. Until, without any preliminaries, we read that on April 7, 1892, thirteen physicians were admitted to membership and that seven of these were women. Their names were Marie K. Formad, Ida E. Richardson, Clara Marshall, Marie B. Werner, Clara Dercum, Anna E. Broomall and Anna M. Fullerton. All were graduates of the Woman's Medical College. Their one time champion, Dr. Albert H. Smith, had died in 1885, and gone to his reward. Their one time opponent, Dr. William Goodell, was still an active member of the Society and recorded as among those present at this memorable meeting.

In 1958, women take an active part in the business and scientific programs of the Society. In 1943, through the efforts of Dr. Louis C. Scheffey and Dr. Charles A. Behney, I was honored by being elected as its President.

It is not necessary to remind this audience that the College of Physicians of Philadelphia is an ancient and honorable organization, founded in 1787. For one hundred and forty-five years it existed without the benefit of female membership. In 1929 two friends of the Woman's Medical College, the late Dr. James M. Anders and the late Dr. L. Napoleon Boston, decided that the Woman's Medical College should be represented in the membership of the College of Physicians. The matter was referred to a Philadelphia lawyer who reported after one year that the constitution of the College would so permit. This being settled, the members spent another year in deciding whether they wished to admit women to membership. Then, since the constitution permitted and since the members wished to do so, who should the woman be? Dr. Martha Tracy, Dean of the Woman's Medical College was consulted and very kindly recommended me. After one more year I was elected.

On the evening of January 11, 1932, in this same Mitchell Hall, with Dr. S. Weir Mitchell looking down from his portrait at the end of the room, I was admitted to membership. The President of the College, Dr. Francis R. Packard, eminent otolaryngologist and medical historian, reached over to shake my hand and greeted me with the words "You are making history tonight, Dr. Macfarlane."

The following year Dr. Tracy herself was admitted to membership.

The College now numbers 1,144 members; of these 42 are women. They take part in scientific and business meetings and serve on committees. The head of the Section on Preventive Medicine was for several years a woman, Dr. Katharine R. Boucot. She is now a member of the Library Committee of the College.

In this room at a meeting of the College in October 1957, eleven new members were admitted; eight of these were men, three

were women. In the words of Galileo "The world moves."

The American College of Surgeons was founded in Chicago in 1913. One of the leaders of the founding group was Dr. Maurice Richardson of Boston. Upon his recommendation, Dr. Florence Duckering, a surgeon on the staff of the New England Hospital for Women was admitted to membership—the first woman to be so honored. Also in 1913, upon recommendation of the Pennsylvania members of the Board of Regents, Dr. Caroline M. Purnell, Dr. Marie K. Formad and I were admitted to membership. We were welcomed on the flag and palm decked stage of the Academy of Music by the President of the College, Dr. William J. Mayo. It was a great moment.

Dr. Mary P. S. Rupert, Woman's Medical College '04, was a charter member of the American College of Physicians. Dr. Alma Dea Morani, Woman's Medical College '31, was the first woman admitted to the Society of Plastic Surgeons. Three graduates of the Woman's Medical College, Dr. Helen M. Angelucci, Dr. Ruth Ellis Lesh and Dr. Alma Dea Morani, were among the first woman to be admitted to membership in the International College of Surgeons.

In 1915, Dr. Bertha Van Hoosen, an able woman surgeon in Chicago, decided that while women physicians enjoyed membership in the American Medical Association and benefitted by the scientific papers and discussions, they were somewhat lost in the overwhelming number of men physicians. They had very little opportunity to become acquainted with each other and returned to their homes without the personal contacts that are such an important feature of medical meetings. To meet this need she organized the American Medical Women's Association. This meets on the two days preceding the annual meeting of the American Medical Association. Membership is limited to members of the American Medical Association in good standing. Its present membership is 2000.

When the United States joined the Allies in World War I, Dr. Van Hoosen appointed a War Service Committee with a very able woman, Dr. Esther Pohl Lovejoy, as its Chairman. Under Dr. Lovejoy's leadership hospitals were established in war-torn Europe. In these the sick and injured were cared for by women physicians whose services were not accepted by the United States Government. This committee has broadened its scope and continues to render valuable service in such widely scattered parts of the earth as Greece, Korea, Haiti, and our Southern Mountains.

On Sunday, December 7, 1941, the Board of the American Medical Women's Association met in Philadelphia at the Woman's Medical College. The terrifying news of the attack on Pearl Harbor came over the radio. It was voted unanimously to send petitions to President Franklin D. Roosevelt, to Secretary of the Army Stimson, and to Secretary of the Navy Knox. Worded by the President of the Association, Dr. Emily Dunning Barringer, these stressed the need of more physicians in the great emergency confronting the nation and urged the appointing of women physicians to the Armed Services. The voice of organized medical women carried weight. Soon women physicians were admitted to the Armed Services with officer status, not as "Contract Surgeons" as heretofore.

After this brief review of the sometimes uphill road traversed by women physicians over the past 108 years, I believe you will agree with me that we have endeavored, in the words of S. Weir Mitchell, to "repay opportunity with labor."

The Evolution of Plastic Surgery[1]

By ALMA DEA MORANI, M.D.[2]

PLASTIC surgery as a specialty hardly existed before the First World War, when the need for skillful repair of maxillo-facial injuries focused attention upon it. Yet plastic surgery has been practiced from very early times in various parts of the world, and history reveals its progress has been strangely intermittent. For long periods this surgical art has been neglected and knowledge of its principles and techniques almost forgotten, yet at intervals there has been an awakening of interest, sometimes in response to a particular need, sometimes apparently as the result of chance.

The earliest definitely recorded development was in answer to a widespread demand. It occurred in India approximately 2000 B.C. when nose amputation was a frequent punishment for criminals, prisoners, and unfaithful wives. Split earlobes were also common, and various mutilations of the face and sex organs were often practiced to disfigure an enemy. It is therefore not surprising that Hindu surgeons began crude attempts to repair such mutilations at an early date. Nasal reconstruction utilizing a flap from the cheek was described by Sushruta, the Father of Hindu Surgery (about 750 B.C.). He also described a method of repairing injured lips by means of cheek flaps, and enumerated fifteen ways of dealing with mutilated ears. The practice of featural restorations became a prominent part of the medical writings of that period.

The first European writer to mention plastic operations was Aurelius Cornelius Celsus (53 B.C.–7 A.D.), who lived in the time of Tiberius Caesar. Celsus gave detailed accounts of many techniques to be employed for repairing loss of tissue of the nose, lips or ears.

History relates that Justinian II (669–711 A.D.), Roman Emperor, was so harsh a ruler that his subjects rose in rebellion, took the Emperor prisoner, cut off his nose, and sent him into exile. Later he escaped, raised an army, recaptured Constantinople and again ascended the throne. Naturally the Emperor demanded a nasal reconstruction. Today, still preserved in Venice, Italy, there is a marble bust of Justinian showing him with a new nose which had evidently been constructed from a forehead flap. Yet after this triumph on the face of so exalted a personage, the whole art of facial reconstruction disappeared from recorded history for more than seven hundred years. When it reappeared, it was a secret in the hands of a family of surgeons practicing in Italy—the Branca Family of Sicily.

Gaspare Tagliacozzi

The 16th century produced the man who may be regarded as the founder of modern plastic surgery. Gaspare Tagliacozzi (1545–1599), professor of surgery at the University of Bologna, used a flap from the arm for nasal reconstruction, a technique now known as the Italian Method of Rhinoplasty. Tagliacozzi devised many other plastic operations and in 1597 wrote the first systematic treatise on plastic surgery, *De Curtorum Chirurgia per Insitionem.*

In October 1794, a new interest in reparative surgery was inspired from a completely unexpected quarter. A London magazine carried a report of a Hindu prisoner whose nose had been amputated and a new nose successfully reconstructed. The surgeon was a man of the brickmaker cast who used a forehead flap for this reconstruction. Considering the method used (no lining of skin flaps) it is doubtful that the nose was a functional and cosmetic

[1] Read before the Section on Medical History, College of Physicians of Philadelphia, 21 January 1958.

[2] Clinical Professor of Surgery, Woman's Medical College of Pennsylvania, Philadelphia 29, Pa.

FIG. 1. Statue of Gaspare Tagliacozzi, pioneer in plastic surgery, holding human nose in left hand. Tagliacozzi was largely responsible for popularizing the 'Italian Method' of rhinoplasty.

success, but the case report served to stir up the interest of British surgeons in India, and to this day the method is referred to as the Indian Method of Rhinoplasty.

Another pioneer who contributed to the evolution of plastic surgery was Johann Friedrich Dieffenbach (1792–1847) of Germany, who performed his first rhinoplasty in 1829. He was soon performing the operation frequently, as well as reconstructing lips and eyelids. Dieffenbach possessed an engaging personality, was an excellent artist, writer and teacher, and his work was received with enthusiasm not only by the medical profession but by the public and the press.

The 19th century produced many British and American surgeons who did valuable original work in plastic surgery. Briefest mention of these men would include Dr. J. Mason Warren (1811–1867) of Boston who performed the first rhinoplasty done in this country, using a forehead flap. He was also successful in the use of free full thickness grafts for repairs on eyelids, lips and noses. John Peter Mettauer (1787–1857), contributed to surgical treatment of cleft palate. Thomas D. Mütter (1811–1859) of Philadelphia pioneered in the treatment of burn contractures and deformities and originated methods for their relief by skin grafts and pedicle flaps.

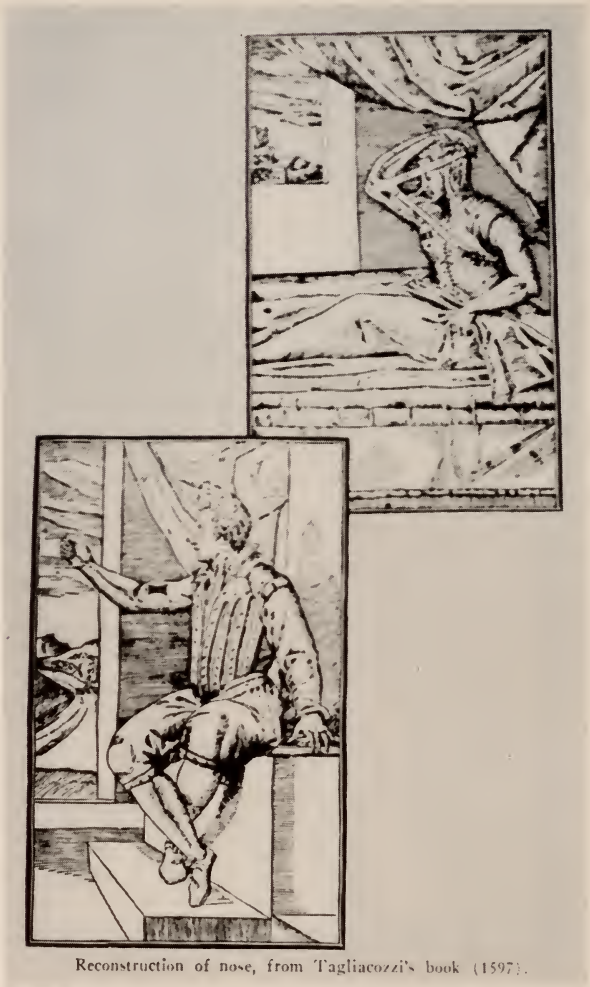

Fig. 2. Diagrammatic drawings showing the 'Italian Method' of rhinoplasty.

During these same years many new developments in skin grafting were offered in Europe by J. L. Reverdin (1842–1929), E. L. Olier (1825–1900), Karl Thiersch (1822–1895), John Reissberg Wolfe (1824–1904), and Fedor Krause (1857–1937). As time went on it became apparent that there was need for the transplantation of tissues other than skin, such as bone, cartilage, muscle, nerve, fat, mucous membrane and cornea.

With the First World War came the beginning of a new epoch in this surgical specialty for surgeons of the Allied Forces early recognized the desirability of training surgeons in the treatment of various maxillo-facial wounds. Both Britain and the United States ordered general surgeons, oral surgeons, rhinologists, dental surgeons, ophthalmologists and neurosurgeons to form teams that could develop the field of plastic surgery. It has been aptly stated that plastic surgery became fathered by specialists in all the most intricate and difficult forms of head surgery, while the mother of plastic surgery we can consider to be "ingenuity".

As late as 1916 no separate department of plastic surgery existed in any medical school or hospital in America. Today, however, there are well organized departments of plastic surgery

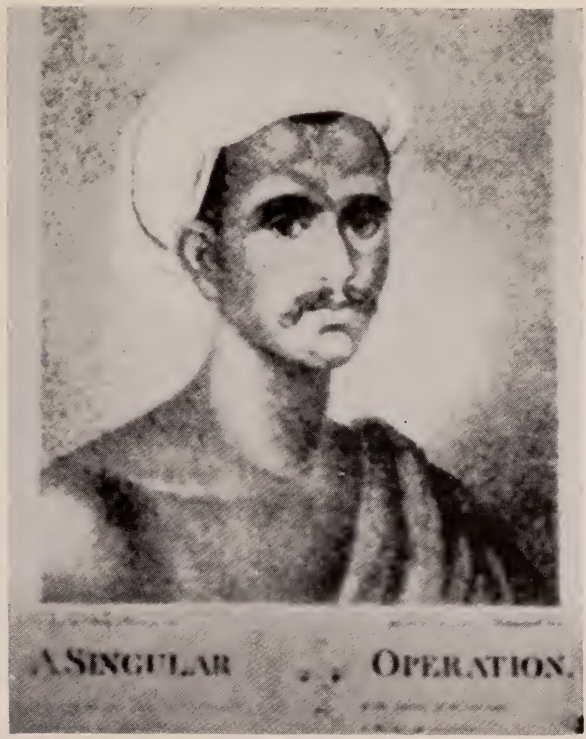

FIG. 3. Frontispiece from a British Magazine (1794) describing the 'Indian Method' of rhinoplasty.

in the majority of medical schools and large hospitals.

With the advent of World War II, many plastic surgeons were available and a general recognition of the importance of their work existed. In England, nine centers were created for maxillo-facial cases, largely through the direction of Sir Harold Gillies, who exercised his unique creative talents in restoring disfigured parts to normalcy. Queen Victoria Cottage Hospital in East Grinstead, England, saw more than 7000 operations performed before the end of 1943, and it was here that Gillies earned the reputation of being the outstanding plastic surgeon of this century. In America, six of the army's large general hospitals were designated for soldiers requiring plastic work and were specially staffed and equipped to that end.

Thus it was that the experience gained by plastic surgeons in two world wars taught the medical profession a great many valuable lessons. Fundamental plans of treatment were correspondingly soon established for the repair of congenital and traumatic deformities. Great refinements in technique have since been made possible; the dermatome was a particularly great contribution to the treatment of burns.

These wartime advancements, moreover, were translated into use for the benefit of the injured civilian. When the mechanical age produced as many civilian casualties as the previous wars, the need for plastic surgery as a separate specialty was definitely established. Perhaps no field in medicine has shown greater strides in the last decade for because of military plastic surgery, civilian plastic surgery has increased in scope and demand. New methods and ideas are constantly being tried with the artistic zeal common to plastic surgeons. Today few cases occur in which a satisfactory reconstruction is not possible, and for such cases prosthetic appliances are employed.

The principles and art of plastic surgery are used today by reconstructive surgeons who are capable of invading every area of the human

body, whose field encompasses not only the repair of trauma of war or peace, but the reconstruction of congenital defects and even a consideration for cosmetic improvements.

Some observations on plastic surgery of this decade remind us that great surgical figures were involved, names like those of Vilsay B. Blair, John Staige Davis, Victor Vean, and Morestin emerged from many surgical fields to devote themselves to this new camp. These men sought their training by traveling widely and frequently quarreling with each other, but they never ceased to teach and spread the gospel of plastic surgery. To them and to Dr. Robert H. Ivy of Philadelphia we all owe a debt of gratitude. For these pioneers bequeathed to this specialty the architectural principles upon which plastic surgery is based, and went on to create plastic surgery societies with a broad virile outlook of what these reparative and reconstructive fields should encompass.

Advances in general surgery obviously helped make possible many plastic procedures previously unsuccessful. I refer to the use of blood and plasma transfusions, tetanus antitoxin, anti-shock therapy, and chemotherapeutic treatment to combat infections. These measures, with the understanding of good fluid and electrolyte balance and modern anesthesia, permitted plastic surgeons to expand their art to include operations of considerable magnitude. Recently, the antibiotics have added another victory over infection and further contributed to the huge area of tissue grafting.

The story of plastic surgery has evolved slowly in the past and any attempt to forecast its future might be considered fantastic. There are many indications, however, that the scope of plastic surgery today is so wide that the transplantation of entire organs or parts will soon become realistically possible.

The history of this surgical art is one of splendid achievement in the past and a glowing vista ahead. Today the plastic surgeon is everyman's surgeon.

I would conclude this brief review of the evolution of plastic surgery with the words of Tagliacozzi who, in 1570, wrote—"We restore, repair and make whole those parts which nature hath given, but which fortune has taken away, not so much that they may delight the eye, but that they may buoy up the spirit and help the mind of the afflicted".

Science and Sentiment: A Study of Oliver Wendell Holmes*

By ELEANOR M. TILTON, Ph.D.†

TO TALK about Dr. Oliver Wendell Holmes seems supererogatory; for the doctor talked well and often in fact and fiction, and always essentially his subject was himself. The severest of his medical essays are not untouched by that conceit of his own cleverness that amused some of his contemporaries and irritated others. The members of his family did not go out in public with him without a twinge of apprehension lest father show off. Even his mother good-humoredly recognized his foibles; about to visit the doctor and his bride and writing them both, she addressed an aside to her daughter-in-law: "We will release the Doctor & have a chat to ourselves if you please for I feel constantly as if he was prepared for the lecture room . . . ."[1] He awoke in his contemporaries every degree of response from wrathful scorn to enthusiastic affection. Offended by his strictures on Copperheads in his Fourth of July Oration of 1863, an irate victim wrote nastily: "As for Holmes Senior, I agree with you fully that he is a miserable little mannikin, dried up morally and physically, and there is certainly nothing more aggravating than to have such a little fool make orations . . . ."[2] At the opposite extreme is the record of Alice James, sister of the novelist, whose diary notes:

> What a delightful, generous, and human sound Dr. Holmes' letters have, especially in contrast to the self-conscious Lowell. It makes one understand father's enthusiasm for the Doctor, who, he used to say, was worth all the men in the club put together; and how indignant he used to be with Lowell's manner of snubbing him, and admiring of the perfect way the Doctor took it.[3]

For Henry James, the elder, father of the novelist and of the philosopher William James, Holmes was more than the equal of the distinguished members of the Saturday Club, that is Agassiz, Lowell, Longfellow, Dana, Hawthorne, Motley, and Emerson. We can let Emerson strike a balance between these extremes of judgment; he once observed: "Well, he talks a great deal, but Holmes has great rights."[4]

Holmes's daughter Amelia leaves us a half-proud and half-embarrassed account of the candid doctor exercising those rights:

> I have now only one page left to describe to you a great spree that Pa & I went on last night. Mrs. Mountjoy invited us & others to her house last night to see the "Spirits." They consisted of a showman & his daughter of 13—She was tied tight & put into an empty bathing house (or what looked like one) & there she sat while a deceased spirit talked & musiced & did many other things. At least that was what the showman said it was. But before we began Father & Dr. Warren were appointed the committee to

* Kate Hurd Mead Class of 1888 Lecture in Medical History (X), Woman's Medical College of Pennsylvania, College of Physicians of Philadelphia, Section on Medical History, 15 April 1958.

† Associate Professor of English, Barnard College, Columbia University, New York 27, N. Y.

[1] A.l.s., Sarah Wendell to Dr. and Mrs. Holmes, Sept. 28 1840; Harvard Holmes Collection, hereafter referred to as HHC.

[2] M. deW. Howe, *Justice Oliver Wendell Holmes* (Cambridge, 1957), I, 145.

[3] A. James, *Her Brothers Her Journal*. ed. by Anna R. Burr (New York, 1934), p. 122. The letters referred to are those to John Lothrop Motley, written during the Civil War.

[4] *The Christian Register*, June 7, 1883, p. 360. Emerson is quoted by Frederick Henry Hedge; the occasion is the annual Unitarian Festival, at which Holmes presided; Hedge is replying to Holmes's introduction of him.

> examine. And of course my pa was the spokesman—And what did he say when asked at the end by the showman for his report. Why he got up & in the brassiest yet very blandest manner in most picked terms announced that he thought it was a very fine piece of jigglery & manipulation. Of course he set all believers by their hair & ears. And five or six of such instantly all at once began upon him. But he with his usual brass upon such occasions held his ground manfully, & six fifths of the audience being on his side, the eloquent orator was firmly supported. I staid [*sic*] in one red blush all the time father was pitching into them, & I thought what Mother would have done if she'd been there. She was not invited but she thought she should like to have gone till she heard my report of fathers [*sic*] proceedings.[5]

Characteristically here, the doctor's sense of public sentiment, which served him so well when he had to provide verses for civic occasions, failed him as the scientific observer took over.

The "fatal facility" for utterance in talk, prose, and verse was not always to Holmes's advantage; it was never less so than when the matter in hand appealed to the autocratic professor of anatomy rather than to the sentimental poet. In his letters and in his talk, he was likely to be even less considerate than in print of people's emotional attachment to their erroneous religious or medical beliefs. Anyone hinting at acceptance of homeopathic doctrines, for example, was likely to get short shrift from the doctor; one such speaking favorably of homeopathy provoked the retort: "I haven't much respect for your intelligence."[6] To the Reverend Theodore Parker, who had admitted that his doctor inclined to homeopathic practices, Holmes wrote: "The truth is, I suspect, that about one fourth part of quackery to three of science is the thing for a doctor, as one fourth of superstition to three parts of theology makes on the whole the best priest."[7] Wherever science directly or indirectly overrode sentiment, Holmes might find himself, sometimes to his wounded surprise, in hot water. He appears never to have observed that his scientific training, such as it was in his day, consorted ill with his poetic inclinations, but he was aware of the reverse. Answering a young stranger's query about the wisdom of combining a medical and a literary career, he wrote: "Medicine is very exacting. I don't believe much in literary doctors. I would not have one that was in the habit of scribbling verses or stories or anything of the kind."[8]

That Holmes chose as his profession medicine rather than law or theology, that he studied in France rather than in Germany, and that his principal subject was anatomy rather than metaphysics—these experiences gave him an intellectual orientation different at every point from that of his contemporaries who were also poets, lecturers, and writers of fiction. As a writer Holmes liked to offer himself as an amateur: he was anxious to let Emerson know that his kind of lecture was not in the same class as Emerson's, to let Lowell know that he had not Lowell's scholarly knowledge of the literature of the past, to let everyone know that his novels were such as anyone might write, and to pass off his talent for verse as an infectious disease. These self-protective devices suggest that Holmes was nervously aware of being differently directed from his contemporaries. In one of his numerous attempts to establish the fact of human limitations, he said that "a man is not answerable for his congenital dispositions, his education, his particular gamut of powers and sensibilities."[9] One might

[5] A.l.s., Amelia Holmes to O. W. Holmes, Jr., May 8, 1866; original in the Harvard Law Library, quoted in T. F. Currier, *A Bibliography of Oliver Wendell Holmes*, ed. by E. M. Tilton (New York, 1953), p. 488, hereafter referred to as *Bibliography*.

[6] F. H. Hedge in response to Holmes's introduction of him cited above, note 4.

[7] A.l.s., Holmes to Parker, Sept. 14, 1857; no. 79 in bound volume of letters, HHC.

[8] A.l.s., Holmes to "My dear young friend," Nov. 12, 1865; recently added to HHC.

[9] Ms. notes for the essay "Crime and Automatism" in Commonplace Book (contains notes for *Over the*

suppose that education would be considered as within one's control; Holmes appears to have seen that science separated him from his fellow literati. His critics told him plainly that he was off side and frequently did so with reference to his profession, accounting for his deficiencies on the grounds that he was a doctor. Certainly he did not march to the same tune as many of his contemporaries.

He was out of step with the humanitarian reformers of his day, with those who were trying to rescue religion from Calvinism on the one hand and Unitarianism on the other, and with those who were probing the reaches of the soul in search of absolute Truth. In consequence the literary historian has some difficulty in assigning to Holmes a place and may be tempted to regard him as a vestigial remain of the eighteenth century or a forerunner of the twentieth. One has no difficulty at all in tying neatly the threads that bind Longfellow, Whittier, Emerson, Hawthorne, and Poe together and then extending the net to catch the younger men, Thoreau, Lowell, Whitman, and Melville. The flexible rubic "romantic" covers them all, but does not touch Holmes; they do not all represent the same kind of romanticism, but they can be brought together on that high and varied plane.[10] Searching to define Holmes's place, we may glance briefly at where he stood in relation to his fellows on three matters of concern in his times: reform, religion, and psychology.

Holmes's refusal to join with the humanitarian reformers in their large undertakings might seem to put him in Emerson's camp; but he was not at the time aware of having any such ally as Emerson. When he came to write Emerson's biography, he took a somewhat guilty interest in discovering that Emerson had had no truck with the abolitionists, apparently finding in the fact a retroactive justification for his own stand, for which he had been roundly abused, and forgetting that among the private critics of his New England Society address of 1855 had been Emerson himself. Regarding the reformers as possessed of an imperfect intuition of the True Good, Emerson did not care to join them, but he did not approve of any word or action that might stand in their way. On this high ground Emerson could stand aloof from reform groups at the same time that he could indirectly rebuke Holmes for lightly criticizing such groups.[11]

The reformers themselves, not infrequently misrepresenting Holmes's words, expressed their exasperation with him in public print and by private letter. He ought he was told to use his gift for words in the just causes of temperance, peace, and abolition. Having no reason to suppose himself deficient in human and humane feeling, Holmes was both astounded and wounded by the attacks upon him and gave expression to his pained surprise in the verses now titled "The Moral Bully," in letters to his private critics, and in the lecture "Literary Tribunals" (later called "Critics and Criticism") in which he recognized the role of irresponsible journalists. The doctor's accusers, unfamiliar with his professional life, could not know of his vigorous letter to the managers of the Boston Dispensary proposing reforms for that ill-organized medical charity.[12] However notorious his two essays on puerperal fever were among physicians, laymen could not have

Teacups), p. 5; HHC. The allusion to "congenital dispositions" is also personal; see letters by Holmes to S. Weir Mitchell in my *Amiable Autocrat* (New York, 1947), pp. 311–2.

[10] The inclusion of Melville's name might be objected to; certainly he bent much of his energy to hurling thunderous *No*'s in the direction of the romantic idealists, but he was none the less deeply attracted to them and had the attitude of a man who would very much like to be convinced.

[11] See *Amiable Autocrat* for an account of these attacks, pp. 224–8. Holmes got wind of Emerson's criticism through the chemist Dr. Charles Jackson, a relative of Emerson's wife and the disaffected associate of W. T. G. Morton, usually credited with the discovery of ether anesthesia. Although in 1864 Holmes wrote Emerson (a.l.s., March 27, HHC), that he had never been reproached by Jackson for his stand when asked to give his opinion in the ether controversy, I suspect that some rankling bitterness may have prompted Jackson to play the role of tale-bearer here.

[12] The letter was printed in 1857 in a history of the Dispensary (by W. R. Lawrence), but the book was not the sort to have been generally read.

known of the humanitarian impulses they revealed.[13] In the interests of humanity as well as reason, Holmes was prepared to attack in and out of season all varieties of quackery, even the variety his profession is slow to acknowledge as such—the over-dosing of patients. It was not generally known that from first to last Holmes had proposed and voted for the admission of women to the Harvard Medical School, being more often than not the solitary voice supporting their claim.

The humanitarian causes Dr. Holmes was not prepared to support and those that found him ready and eloquent are suggestively different. He was ready to try to right a specific and reachable wrong about which something might be immediately done; and in such instances he addressed himself to those who were in a position to effect the desired change. He was not prepared to tackle a large, general, or remote evil. He was not prepared to prescribe for a complicated social ailment, the causes and nature of which are not intelligible even now. Nor did he care much for the humorless agitator who supposed that men could be browbeaten or exhorted into virtue. It was Hawthorne who observed of reformers that they had no imagination; Holmes might well have made the same remark, for he noted as a defect of reformers that they were incapable of being bored.[14] The reformer's single-minded embrace of a cause was to Holmes evidence of disease. As he wrote in 1850, calling to his aid a famous physiologist:

> No life worth naming ever comes to good
> If always nourished on the self-same food;
> The creeping mite may live so if he please,
> And feed on Stilton till he turns to cheese,
> But cool Magendie proves beyond a doubt,
> If mammals try it, that their eyes drop out.
>
> No reasoning natures find it safe to feed,
> For their sole diet, on a single creed;
> It spoils their eyeballs while it spares their tongues,
> And starves the heart to feed the noisy lungs.[15]

The reformers, who might have lost their vision, had not lost their voice; and the enthusiasm with which they used it betrayed to the conservative doctor the signs of fanaticism. Considering "conservatism as primarily based on a proper recognition of human limitations,"[16] we are bound to label Holmes conservative, for the dearest weapon in the arsenal he turned upon orthodox religion was his demonstration of human limitations.

Holmes first formulated his idea of the limited will in *The Professor*, its formulation forced upon him by the running comment of an irate religious press. The notion itself was probably long in his mind; his ten-year experience as a physician would have brought dramatically before him the fact that men cannot always be or do what they wish. Holmes said as much in a passage prepared for an after-dinner speech for the annual meeting of the Massachusetts Medical Society in 1867. Here he described the case of Mrytle Hazard, heroine of *The Guardian Angel*, then running in *The Atlantic Monthly*. After alluding to the hypothetical and improbable case of Elsie Venner, he says of the second novel:

> Here we come upon the ground of direct observation. I describe the facts of inherited qualities as we who study families see them. I carry my subject through such exposures as our experience of life has shown us to be of too frequent occurrence. I hope to find, as I have found heretofore, many in my own profession who fully recognize the truth of the general laws I illustrate.[17]

[13] Neither the 1843 nor the 1855 printing was a large one. See *Bibliography*, pp. 33–5, and *Amiable Autocrat*, pp. 169–76.

[14] Letter to James Freeman Clarke, Oct. 24, 1862; J. T. Morse, Jr., *Life and Letters* (Boston, 1896) I, 306.

[15] "The Mind's Diet," (originally in *Astræa*), *The Complete Poetical Works*, Cambridge Edition (Boston, 1895), p. 85; hereafter CAM.

[16] Sir Lewis Namier, *Vanished Supremacies* (London, 1958), p. 13.

[17] Strip of newspaper proof (*Boston Daily Advertiser*), pasted in notebook (HHC) for *The Guardian Angel* with ms. note: "Mass Med Soc Left this all out from my dinner speech." The passage does not appear in the *Advertiser* report of the speech, June 6, 1867.

The raisonneur of the first "medicated" novel, *Elsie Venner*, is a doctor whose office is to comment on Elsie not as a medical phenomenon but as an illustration of the fact of human limitation. Much of the chatter of the Breakfast-Table books and all three of the novels are directed to defining and illustrating his notion of a limited will, repudiating on the one hand Calvinistic insistence on original sin and on the other the Arminian notion of freedom of the will. Taking this middle ground, the doctor would be unable to conceive of the possibility of the wholesale reforms envisioned by the zealots for the causes of temperance, peace, and abolition. The possibility that men would voluntarily abandon strong drink, warfare, and slave-owning was to Holmes as remote as the possibility that real Simon Legrees might be visited by irresistible grace and become "saints" like Uncle Tom. Absolute sin and absolute freedom were beyond the reach of the doctor's imagination. As he remarked in a note he cared "nothing philosophically much empirically."[18] The empirical evidence collected from the bedside had helped him a little distance from the Calvinism in which he had been bred but had not brought him into the fold of the romantic idealists of his day.

His romantic contemporaries were accustomed to think on a large scale, to deal in absolutes, and to reject any notion of limitation. Even Hawthorne, who had not the presumption of Emerson or Whitman, dealt with what he called "the Truth of the Human Heart," a subject considerably grander than his small-scale treatment of it. With his eye on the immediate, upon what could come within the range of actual vision, upon what was subject to measurement, Holmes struck his German-indoctrinated contemporaries as myopic. He was a man of mere "understanding," not a man of "imagination." When he treated of certain subjects sacred to the romantic taste; e.g., Carlyle or Wordsworth, he was told that he ought to treat of others in which he would be more "at home." Old admirers of Emerson were outraged when Holmes was chosen and agreed to write, for the American Men of Letters series, a biography of the transcendental sage; here clearly was a subject in which a doctor, a "materialist," possibly even an atheist, and a man of mere "understanding," could not be "at home." Nor were his critics altogether mistaken. That in heyday of transcendentalism, in the 1830's and 1840's, Holmes had known little of the group that had been attracted to Emerson is demonstrated by his not having consulted Frederick Henry Hedge, the prime mover behind the "Transcendental Club," usually known at the time as "Hedge's Club." Candidly and shamelessly, Holmes replied to Hedge's letter of complaint of his having been neglected; the doctor admitted that he had not known that Hedge and Emerson were even acquainted.[19]

Although after his return from France in 1835, Holmes could not have been and was not wholly unaware of what his romantic contemporaries were doing, he remained for the most part unmoved by what moved them—the romantic literature of Germany and the romantic poets of England. That he should have been asked in 1852 to give a series of lectures on the English romantic poets must have seemed an odd choice; and there is some evidence that critics already committed to romantic views were prepared to let fly the moment the doctor opened his mouth.[20] His sudden plunge, at the age of 44, into the works of the romantic poets could not turn him into an idealist, nor change his taste for that poetry that deals with the visible world and with men as they act or feel in response to something outside themselves, nor lead him to abandon the heroic couplet for blank verse. Holmes wrote only one romantic poem—"The Chambered Nautilus," and this production came as a surprise even to its author, who seems to have valued it partly because he could not account for it.

[18] Ms. note, black notebook, p. 124; HHC.

[19] A.ll.s., Hedge to Holmes, Dec. 22, 1884; Holmes to Hedge, Dec. 23; HHC, but see note 4.

[20] For example, *The Commonwealth*, whose reviewer was Julia Ward Howe, was alert to find fault and sharp in doing so.

Hastily providing himself, by purchase and borrowing, with the works of romantic poets and critics, Holmes prepared his Lowell lectures as best he could. He was not wholly unaffected by what he there encountered nor untouched by the criticism his lectures provoked. Both from preparing the lectures and receiving gratuitous instruction afterwards, the doctor could scarcely escape becoming aware that romantic poetry was not the same as that he had become accustomed to in his father's volumes of Pope and Dryden or as that he wrote himself. These romantic poets were personal in a way more intimate than he had ever thought of being, in spite of his inclination to be autobiographical; they were writing about spiritual states and experiences and began not with observations of things and actions but with observations of states of mind. They were, in a word, "psychological," in the romantic sense.

Hawthorne had described his work as "psychological," apologizing for the neologism; Poe might well have called his work "psychological" also; both could have found the word in Coleridge. But the meaning of the word in the romantic sense would not be applicable to Holmes. The work of Poe and Hawthorne is "psychological" because it is about the soul in its traditional sense of that entity that is immortal, that survives dissolution and consequently can carry on colloquies in heaven as in the stories of Poe, that can be saved in spite of sin, and that finally is symbolized by the butterfly as Hawthorne uses it in "The Artist of the Beautiful." In the work of both, details of physical appearance and of décor are employed as symbols or images of the state of the soul that is the subject of the work; Hawthorne found Lavater's notions of physiognomy useful for this purpose. Neither writer is implying any belief in there being a reciprocal relation between body and mind; the relationship, where any at all is implied, is strictly a one-way affair, the soul being the active agent, the body being merely the plastic matter upon which the soul works, works entirely for the artist's convenience. Nor is the soul represented as subject to any cramping limitations, particularly in the work of Poe, who, fortunately for his work, did not suppose that the business of the artist is the presentation of verifiable truth. Poe was, moreover, primarily interested in that aspect of the soul we call imagination, the glory of which is its freedom. Hawthorne was primarily interested in that aspect of soul we call conscience, the chief concern of which, unhappily, is sin. Both drew their essential material from observation of the movement of their own minds. Neither had any desire to report observations on the outer world, which was for them, as it was for Emerson and Whitman, a source of supply for images and symbols that were transmuted for whatever service the work at hand required. What Theodore Parker, critical transcendentalist, called the "little walled garden of the mind" is their subject. When Holmes came to take an interest in this garden, as he did in the late 1850's, he entered it by a different gate and looked upon it from a different point of view.

While romantics sought the infinite in their own souls, Holmes had been busy teaching his students the use of the microscope and defending the numerical method of Charles P. A. Louis at meetings of the Boston Society for Medical Improvement. He was teaching the use of a relatively new instrument and a relatively new method for making more precise observations of nature. Late in 1839 at a meeting of his medical club, Holmes was defending William Wood Gerhard's distinction between typhus and typhoid fever;[21] early in May 1840, at a meeting of Hedge's club, the transcendentalists were listening to a paper on the modest subject of "Man in the Ages."[22] When the transcendentalists began to show themselves in print in their periodical *The Dial*, Dr. Holmes took notice of them. In the same year in which he prepared his first essay on puerperal fever, he was moved to a rude poetic attack on the presumption of the transcen-

[21] Ms. records of the Boston Society for Medical Improvement (Boston Medical Library), Nov. 11, 1839; III, 257–8.

[22] R. W. Emerson, *Letters*, ed. R. L. Rusk (New York, 1939), II, 293–4 and note 202.

dental sage who "shouts in Nature's ears." Hailing the transcendentalists as "Deluded infants," the doctor asks: "will they ever know/Some doubts must darken o'er the world below." One might reasonably expect to find an answer to the sane question: Is puerperal fever contagious. But only a romantic "bedlamite" would put conundrums to "Earth the tongueless and the deaf-mute Time!"[23] Again in 1850, Holmes was provoked to instruct the idealist, rather more politely. In *Astræa*, indirectly rebuking those who supposed that intuition at once reveals the One Truth that is All Truth, Holmes expressed an empiricist's belief: "Each truth we conquer spreads the realm of doubt."[24] He cared neither for the absolute assurance of the Calvinist nor for that of the romantic idealist. Where the skeptical Melville attempted to meet the idealists on their own metaphysical ground, Holmes repudiated the aid of metaphysics altogether. No new form of dialectic was to Holmes of any use whatsoever so long as the "Border-Lines" of empirical knowledge were such as to reveal vast areas of ignorance. He delighted to remind members of his own profession of how little they really knew; that nineteenth-century scientific investigation was disclosing so much was for Holmes indication that there was much more to be revealed. Metaphysical systems, like medical and theological systems, were so many One-Hoss Shays bound to collapse in dust.

When he turned his attention to religion, Holmes did so from a point of view that had nothing in common with that of the romantic idealists, who if they were members of the Transcendental Club were bent on seeking a new and solid ground for religious faith. Far from seeking a ground for faith, Holmes was busy cutting the ground from under it. Although he never professed to be an atheist, he was regarded as one, not only by anxious believers of his own generation, but by thoroughgoing skeptics of his son's.[25] Concealing the drift of his thought, he was likely to straddle certain questions with an equivocal answer. In reply to a correspondent's question as to whether or not there is a future life, he answered: "The natural argument seems to me against the supposition."[26] He directed the whole of his attack on religion to one point—the conception of sin. On the grounds that it was the business of "poets to humanize [the] clergy,"[27] he early and late attempted to show them that the idea of sin was as baseless as the idea of phlogiston.[28] The clergyman ought to emulate the chemist and abandon the discredited idea. The idea of sin was to be discredited not by any idealistic Emersonian aphorism: Evil is the absence of Good, but by the observations of the empirical psychologists. To Dr. S. Weir Mitchell he wrote: "I look forward to the day when the *man-crop* and its conditions will be studied and known as well as the cereal crop."[29] Theological anthropology, he said, was as far from reality as a Japanese anatomical figure;[30] what was needed was a new anthropology that should be as well grounded in direct observation as the subject he so lovingly taught. It was not, after all, as one sees here, the poet who was to humanize the clergy but the scientific investigator. In his notes for *The Poet at the Breakfast-Table*, there is an underlined injunction: "*Work along from physical up to moral questions.*"[31] This order is the order of the three "medicated" novels; moral judgments are inadmissible until one has the patient's history.

[23] "An After-Dinner Poem," August 24, 1843, CAM., p. 57.

[24] From passage now titled "Our Limitations," CAM., p. 85.

[25] Henry Adams describes the doctor as talking "mild atheism." The printed text of the letter reads "wild," but that is an unlikely epithet. *Letters . . . 1858–1891*, ed. W. C. Ford (Boston, 1930), p. 288.

[26] A.l.s., to J. W. Riley (?), Dec. 28, 1867, privately owned. The word "straddle" is stolen from O. W. Holmes, Jr., v. Howe, I, 18.

[27] Ms. notes for "The Pulpit and the Pew," p. 21; HHC

[28] *Idem*; this analogy appears in several of the notebooks, HCC.

[29] A.l.s., June 8, 1871.

[30] Ms. notebook for *The Poet*, p. 8; HHC.

[31] *Ibid.*, p. 2.

The humane doctor was not prepared to attack religion on those points that were not damaging, unjust, or cruel. We do not find him troubling himself in his notes or in public print with such questions as the divinity of Christ, the authenticity of the miracles, or the doctrine of immortality. Here science need not trouble itself to quarrel with sentiments that might comfort suffering or elevate moral sensibility. Possibly indicative of Dr. Holmes's private judgment is a passage from Sainte-Beuve, a passage he copied carefully into one of his notebooks:

> Groan over it or not, as we may, Faith has disappeared. Science, let people say what they will, has destroyed it. It is absolutely impossible for vigorous, sensible minds, conversant with history, armed with criticism, studious of the natural sciences, any longer to believe in the old stories and old Bibles. . . . So let us men and women cease to be children as soon as possible; this will be a hard task to a great many women—and to a great many men too. But in the present state of society, herein and nowhere else lie the safety and virility of nations. They will have to choose between Byzantium and true progress.[32]

In *Mechanism in Thought and Morals*, Holmes allowed himself to set the "man of science" against the clergyman, the latter not directly named, and he does so in terms clearly to the advantage of the former.[33]

This 1872 ØBK address throws out in dazzling profusion a stream of analogies and anecdotes, all suggestive and all pointing in the direction of modern experimental psychology and not at all in the direction of Freudian analysis. A consistent vitalist, Holmes had put his faith in the curative powers of nature, to whose care he leaves all three of the patients in his "medicated" novels. Holmes put very little trust in the efficacy of the interfering arts of either the clergyman or the physician and would not I think have been able to take any more stock in the fiction of the Id than he did in the fiction of Sin. Nor did his probings of the soul lead him to discover an infallible inner guide, either the divine Self upon which Emerson exhorted men to rely or the incorruptible conscience that Theodore Parker discovered.

His psychological notions began as a by-product of his persistent attempts to discredit the idea of sin by establishing the fact of limitation; they appear to have continued under the pressure of his son's disapproval of his never having accomplished a really great work. That young man was consumed by a driving ambition that chilled his friends and drove his father into writing some very bad poetry.[34] The younger Holmes did not approve of the way his father wasted his talent upon ephemeral verses, of the way he too readily responded to the public demand for the products of his quick wits. Paradoxically young Holmes's respect for his father's mind appears to be based upon what the father was driven to do with it under pressure of the son's disapproval. The young man's impression of his father as having "the most penetrating mind of all that lot" and as showing "a fertile and suggestive intellect" must date from the time when the father was busy keeping up with the son.[35] Most of the doctor's little notebooks date from 1865–1881, and the subjects and books upon which many of the notes were made were just those with which his son's generation was concerned. He was reading, for example, the new realistic fiction that was so much to influence his son's friends, Henry James and William Dean Howells. The doctor read Turgenev's *Rudin* and could not have failed to recognize in its hero some of the features of his own romantic contemporaries;

[32] Black notebook, p. 61; HHC.

[33] *The Writings*, Riverside Edition (Boston, 1891), VIII, 309–10.

[34] See *Amiable Autocrat*, pp. 309–13; Howe, I, 20, 282. The first and fourth poems, "Ambition" and "Master and Scholar," in the series "Wind-Clouds and Star-Drifts" are certainly the doctor's not altogether comfortable reflections on his son's ambition and their relationship (CAM., pp. 171–2, 174–5).

[35] Howe, I, 19.

he read George Eliot's *Middlemarch* and must have been pleased by its allusions to his old teacher Louis; he read Flaubert and Zola not with moral disapproval but with distaste, coming finally to the conclusion that the only "true realism is that of *Science*. Its distinction is that it is totally indifferent to the aesthetic relation of ideas."[36]

Not himself indifferent to the "aesthetic relation of ideas," Holmes expressed his psychological notions by means of metaphor and simile; on that account, these notions can be made to have any modern look the reader cares to give them. Drawn partly from his reading in contemporary psychology and partly from observation of his own personality, these notions have no ground in investigation and remain what his son rightly called "sporadic aperçus."[37] And always the poet-professor's intention is to demonstrate the limits of will and consequently of responsibility, to demonstrate these limits partly to confound clergyman but also partly to comfort himself. Characteristic of his poetic way of expressing his notions is his use of the image of a compass; a notebook entry reads: "Needle on pivot with no forces acting on it—there is no such condition of the will—no unpolarized state."[38] The same image serves him in his comments on semantics.

That a need for self-justification—a need he recognized—led him to these *aperçus* is illustrated in an unfinished blank verse poem in which a poet who has been exhorted to transcend his accomplishment desperately defends himself:

> Look you, this choosing seems a simple thing
> But could we always choose as we would choose,
> When nothing seems to bind us in our choice
> What painters, sculptors, poets, we might be!

And in lines even worse than these, he goes on unhappily imagining that he is enjoined to produce *Hamlet* or *Paradise Lost*, achievements he knows are far beyond his powers.[39] Reading this desperate and desperately bad effusion, the distant biographer contemplating Oliver Wendell Holmes senior and junior concludes that to be the father of such a son might be more painful than to be the son of such a father.[40] The father attempting to serve two loves, science and sentiment, failed to satisfy the son's lofty notions of achievement; the young man himself dabbled for a time with sentimental verses, but soon turned to devote himself to the profession that not he but his father had chosen for him.

The same letter, already quoted, in which Dr. Holmes advised a prospective doctor not to serve also the muses, has this to say of medicine as a career:

> To be a physician the following requisites if not absolutely necessary are very desirable.
> 1. A sound constitution. The wear and tear are very great, and cares and broken rest and irregular meals and exposures of all kinds demand good stamina.
> 2. An unselfish nature. You must always

[36] Ms. notebook for *Over the Teacups*. p. 19; HHC. In the notebook for *A Mortal Antipathy* (p. 17), he observed of these same realists that it was "time for the decently immoral to interfere with the nauseating realism of Zola and the rest."

[37] Howe, I, 19.

[38] Black notebook, p. 63; HHC.

[39] *Bibliography*, p. 347. The invocation to the stars in the last line of this unfinished poem suggests that it was written at about the same time as the blank verse poems in *The Poet*. The ms. notebook for *The Poet* has a fragmentary statement of the theme: "The will—choose the words of Paradise Lost/You can choose the words of/," p. 9 (HHC). A similar note appears in the earlier notebook for *The Guardian Angel*: "Can *choose* —why not choose the words that make a poem etc." p. 22 (HHC).

[40] The younger Holmes admits to having been "disagreeable," Howe, I, 18, 19. The elder admits to seeking rationalizations for his failure, *Amiable Autocrat*, p. 312. In his copy of Boswell's Life of Johnson, Holmes marked the following characterization of Goldsmith: "He, I am afraid, had no settled system of any sort, so that his conduct must not be strictly scrutinized." Holmes probably recognized the applicability of the judgment to himself. He owned a ten-volume edition of Boswell, edited by J. W. Croker, 1833; the passage marked appears at II, 191; the set is in the Library of Congress.

think of your patient's welfare, not of your own comfort or habits.

3. You must be content to wait a long time before you establish a paying reputation.
4. Much of your work being distasteful, wearisome, wearing to the body and almost fruitless to the mind you must gradually harden yourself to the routine, and for this you need to have an easy and accommodating temper.
5. You must live in constant familiarity with suffering of all kind[s] which must either make your feelings tough or keep you in suffering yourself.

I expect to have both my boys become lawyers.[41]

[41] Italics of original. See note 8.

Primitive Trepanation: The Beginning of Medical History[1]

By A. EARL WALKER, M.D.[2]

IN HIS first lecture on the evolution of modern medicine, given at Yale University in 1913, Osler (3), under the heading of the origin of medicine, briefly refers to "the oldest existing evidence of a very extraordinary practice" in primitive medicine. It is hoped that a survey of this prehistoric surgery may give a perspective to our modern neurosurgical specialty.

Neolithic Cranial Surgery

Before civilization developed in the river valleys of the Nile, Tigris, Euphrates and Indus, from the tenth to the fifth millenium before Christ, neolithic man roamed the western parts of Europe, and buried his dead in dolmens, some of which have withstood the ravages of time. In these graves only one impeccable evidence of prehistoric medical practice is available—namely the presence of holes in the skull, the margins of which have signs of healing.

Like many other discoveries, this evidence was available for some time before it was recognized for what it was. For years holes in neolithic skulls were considered the result of trauma. Such perforated skulls were found in France in the 17th and early 19th centuries and one was illustrated in a book on American skulls published in 1839. In all of these instances it was assumed that the defects were the result of injury. It was not until 1874 that this issue was settled, at which time Prunières (5) presented to the French Association for the Advancement of Science at Lille a detailed account of his studies on this subject and exhibited some dozen skulls having holes, some of which unquestionably had been made during life. The parietal region, particularly the left, was the most common site of the perforation—the occipital and frontal regions were also violated, but the temporal bone rarely. The defect was usually oval in shape, the long axis being in the antero-posterior plane. The size of the cranial dehiscence varied from a few centimeters to almost half the skull; one cranium had a hole 13 x 10 cm. Not all of these holes in the prehistoric skulls were made before death. Some were obviously made after the demise of the individual—whether for the purpose of embalming or of obtaining an amulet or what, remains unknown. But many of the defects had bony proliferation smoothing their margins as if healing had been proceeding for some days or months before death.

The basis of this practice and the techniques employed can only be pure speculation and will not be discussed further. Irrespective of their origin, these prehistoric perforated skulls found in European burial grounds belonging to people living 5–10,000 years before Christ remain the earliest evidence of surgical practice.

Early Peruvian Cranial Surgery

Almost a hundred miles south of Lima along the sandy coast of Peru is a small peninsula jutting into the blue Pacific. Today one may walk along the sandy ridges and kick the dried-out bones and bits of cotton cloth—centuries old—which have become strewn on the gravel. But these now dry, barren, sandy hills rising from the beach, centuries ago were fertile and cultivated by a people of high culture and development. The evidence for this

[1] First Annual Osler Lecture of the Osler Medical History Club of the Jefferson Medical College of Philadelphia, presented 11 March 1958 before the Section on Medical History, College of Physicians of Philadelphia.

[2] Professor and in charge of the Division of Neurological Surgery, Johns Hopkins University School of Medicine, Baltimore 5, Maryland.

comes entirely from the materials found in the burial caverns in this peninsula; no written records are available. Between 1925 and 1930 Professor J. C. Tello excavated the tombs of this peninsula, unearthing archeological material of a civilization not even mentioned by the pre-Columbian writers. The fardels or cloth coffins of these people contained fine wool and cotton material and luxurious clothes, beautifully embroidered in many colors. Pottery, baskets, bone and stone articles indicated a certain degree of industrial development of the inhabitants of this area. Such was the culture of these people. Of medical importance are two findings, namely that almost all of the skulls were deformed and perforated.

Although impeccable evidence of the cranial surgery is available, the means for its accomplishment remains a secret, almost impossible of solution. That the early Peruvians had knowledge of anesthetic or analgesic drugs, access to antiseptic agents and complex surgical instruments can perhaps be inferred from customs and practices apparently handed down through the centuries and from ceramic art. Thus Pardal (4) notes that the Araucanos used the flowers of the Datura Fecox which contains scopolamine, and lesser amounts of hyoscamine and atropine. Many historians of the pre-Columbian era refer to the coca bean as the divine plant of the Incas. Then it has been stated that poultices containing herbs such as coca leaves applied to the head might induce a local anesthesia or analgesia of the scalp. From these facts it is extrapolated that the Peruvians induced general anesthesia by alcoholic drinks or narcotic drugs, with or without the use of some locally acting agent.

Similarly, since there seems to be clear evidence that infection was unusual, it is generally thought that the Paracans used antiseptic agents on their wounds. In the tombs of Paracas, salts of mercury were found which might have been used in aqueous solution or oily suspension for dressing the wounds. Arsenic and copper sulfate also appear to have been known, as well as a number of herbs used for infusions, ointments or tablets for chewing. Certainly the ancient indians of Peru in embalming used substances, rich in cinnamic acid, which have antiseptic properties. Thus it is generally considered that in addition to or in association with his magic incantations, which might have had a hypnotic effect on the patient, the Shaman anointed the site of operation with the water of the God of Health, selected through trial and error for its ability to prevent putrefaction.

There is perhaps more reliable data concerning the surgical instruments, for within the tombs of Paracas were found a number of tools applicable to cranial operations. The tumi, or half-moon cutting instrument could cut the soft tissues of the scalp to the bone. A tumi-chisel of hardened copper gouged out the calvarium in more than 90% of the skulls of Paracas. With this instrument and a little patience a hole of any size could be made in both layers of the skull. In a few instances the defect in the skull was made by drilling a circle of perforations and breaking or cutting the intervening bone.

The control of bleeding must have presented a problem. Digital pressure may have been employed. Possibly a cord tied about the head just above the ears may have been used, as it was somewhat later in Peru. Then, too, it is possible that astringent vegetable products were available. A shrub, pumacucu, common in the Peruvian Andes, has tannic acid as an active ingredient and is used by native surgeons to stop bleeding from wounds.

Needles of bone have been found in Paracas and at least one skull in which sutures were used. Cotton was presumably the suture material, although in one patient, who apparently died shortly after operation, the scalp margins were approximated by tying together groups of hairs from the apposing sides of the wound.

The incision was then dressed with rolls of cotton in the form of a doughnut and the head then covered with rolls of cotton dressing. Numerous examples of such dressings have been found.

In a few skulls the defects were filled with a

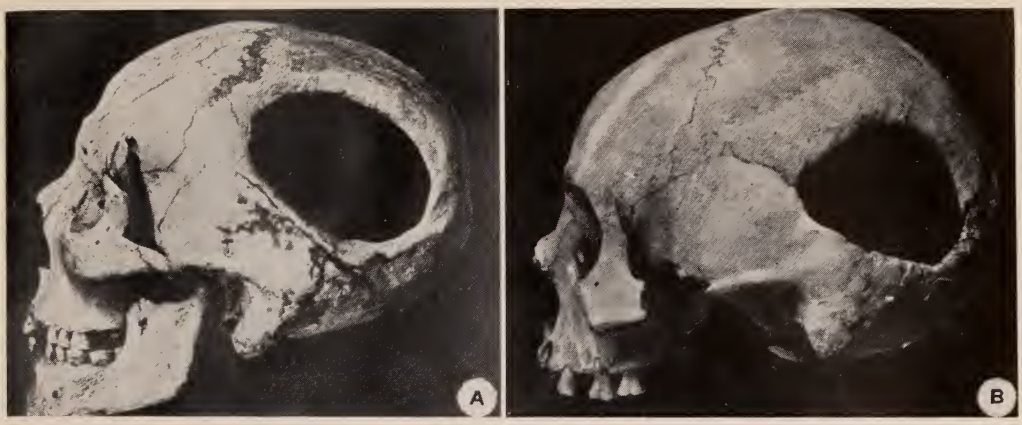

FIG. 1. Examples of cranial defects found in a neolithic skull from France (A) and in a Paracan skull from Peru (B).

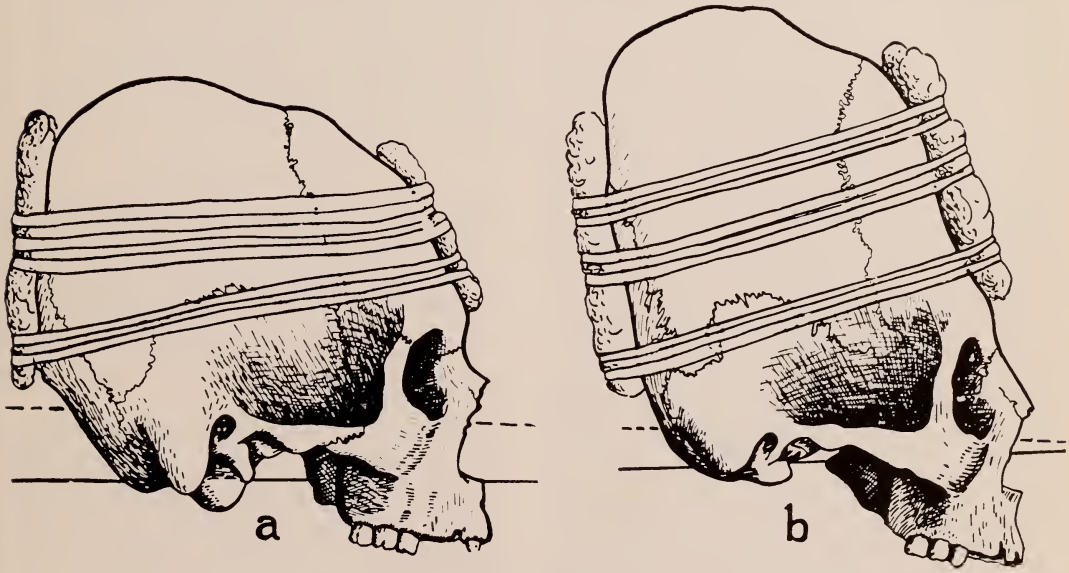

FIG. 2. The appliances found in the fardels of Paracas which were used on babies for artificial frontal (A) and occipital (B) flattening of the skull.

gold or silver plate, but this was the exception in the skulls of Paracas.

The most common type of cranial defect has been circular (fig. 1). In the recent monograph of Graña et al (2), 194 openings in the skull were circular, 26 oval, 40 triangular, 3 quadrangular, 6 rectangular, 4 polygonal and 20 irregular. The location of the cranial defect was much more commonly over the central or parietal region than elsewhere in the skull, although defects have been found in all parts of the convexity. In Graña et al's series the left side was involved in 161 instances, the right in 71 and both sides in 83. Multiple defects were commonly noted—some apparently being made simultaneously and some seriatim, as many as five times. In about a quarter of the cases the cranial perforation was only of the outer table of the skull, the inner table remaining intact. It is noteworthy that males predominated over females 4 to 1.

The mortality of this surgical procedure in Paracas was not excessive. Precise figures based on evidence of bony regeneration are not

available because authors include skulls in their series from other regions operated upon hundreds of years later. The mortality in these reports has varied from 34.5% to 10.5% (6, 7).

The second finding of medical note in the skulls of the caverns of Paracas is the extremely high incidence—as high as 90%—of artificial cranial deformation. The moulding was of either a frontal or occipital flattening, both of which tended to elongate the skull. The mechanics of the deformation are known from the finding of a number of babies' skulls in the caverns of Paracas still in the deforming appliances (fig. 2). It would seem that occipital or occipital and frontal splints were applied to the head in early infancy until the desired contour of the head had been obtained.

The coincidence of artificially deformed heads and cranial trephination in Peru would seem to be not necessarily causally related. It is, of course, tempting to suggest that the cranial deformation caused, not only the bulging of the eyes and distortion of the base of the brain which was known to result from the custom, but also perhaps a herniation of the uncus through the tentorium which might give rise to infarction or compression atrophy of the medial surface of the temporal lobe, resulting in psychomotor epilepsy. In fact, Earle and associates (1) have suggested that the moulding of the skull during fetal life might be a factor producing incisural sclerosis giving rise to convulsive attacks in later life. Attractive as this hypothesis may be, after a critical analysis of all facts there seems to be no basis for the assumption that artificial cranial deformation was in any way causally related to the cranial surgical practices of the people of Paracas.

Then what may be the basis for their extensive cranial surgery? With such a high percentage of the population so involved, one would think primarily that a social custom or religious ritual would be the most probable explanation. If such were the case, did the multiple defects indicate a more exalted person in the social or religious hierarchy?

The cranial surgery of the primitive races, which has been discussed this evening probably is man's oldest and boldest attempt to modify his physical and perhaps mental state to meet his environment more effectively. But the technique of the unknown neolithic and Paracan operators should not be designated as trepanation or trephination, which was to be the next stage in the development of brain surgery. These terms imply the use of a cutting instrument rotating about a center, which required a higher civilization than existed at the time when the first holes were made in the head.

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Transactions of the Sections

SECTION ON GENERAL MEDICINE

24 February 1958

Fatal Typhoid Fever in an Illegal Immigrant: Case Report. Carmen T. Bello, M.D., and Fred B. Rogers, M.D. (Discusser: Angelo M. Perri, M.D., M.P.H.) (*Abstract 1, below*)

The Natural Course of Intermittent Claudication as Determined by Claudication Tests. Raymond Penneys, M.D., and Orville Horwitz, M.D. (Discusser: Walter F. Ballinger*, M.D.) (*Abstract 2, below*)

A New Therapeutic Agent for Acute Nickel Poisoning. F. William Sunderman, M.D., Ph.D., and F. William Sunderman, Jr., M.D. (Discussion: Joseph Seifter, M.D.) (*Abstract 3, below*)

Factors Contributing to Mortality in Experimental Pancreatitis. Alexander W. Ulin, M.D., and Irvin H. Sokolic*, M.D. (Discusser: Thomas E. Machella, M.D.) (*Abstract 4, below*)

24 March 1958

Adrenal Cortical Carcinoma Causing Feminization in an Adult Male: Hormonal Considerations and Heterotransplantation of the Tumor in Guinea Pigs. Lewis C. Mills*, M.D. (Discusser: Eleanor Smith*, M.D.) (*Abstract 5, below*)

Primary Pulmonary Hypertension in Xanthomatous Biliary Cirrhosis. Edward S. McCabe, M.D. (Discusser: Charles M. Thompson, M.D.) (*Abstract 6, below*)

Transplantation of Homologous Thyroid and Parathyroid Glands: Case Reports. Julian A. Sterling, M.D. (Discusser: Edward Rose, M.D.) (*Abstract 7, below*)

Congenital Virilizing Adrenal Hyperplasia in Identical Twins. Norman G. Schneeberg*, M.D., Arthur Steinberg*, M.D., Maurice Malen*, M.D., and Benjamin Chernoff*, M.D. (Discusser: William H. Perloff, M.D.) (*Abstract 8, below*)

26 May 1958

Report of a Case of Nocardiosis due to Nocardia Asteroides Involving the Lungs. Frans J. Barends*, M.D. (Discusser: Edward D. Delamater*, M.D.) (*Abstract 9, below*)

Hemoptysis in Older Men. Katharine R. Boucot, M.D., David A. Cooper, M.D., and William Weiss*, M.D. (Discusser: Martin J. Sokoloff, M.D.) (*Abstract 10, below*)

A Case of Systemic Cryptococcus Infection Treated with Amphotericin B. John G. Rogers*, M.D., and Perry S. MacNeal, M.D. (Discusser: A. Reynolds Crane, M.D.) (*Abstract 11, below*)

Pulmonary Embolism as a Cause of Pleurisy with Effusion. Albert Sheffer*, M.D., Dick D. Harrell*, M.D., and Harold L. Israel, M.D. (Discusser: Julius L. Wilson, M.D.) (*Abstract 12, below*)

SECTION ON MEDICAL HISTORY

11 March 1958

Meeting sponsored by the Jefferson Medical College of Philadelphia. Lewis C. Scheffey, M.D. presiding.

Primitive Trepanation: The Beginning of Medical History[1]. A. Earl Walker*, M.D. (First Annual Osler Lecture, Osler Medical History Club, Jefferson Medical College; Lecturer introduced by David J. LaFia, M.D., Faculty Sponsor).

W. W. Keen's First Modern Brain Case. Donald S. Willard*, A.B.

Some Personal Reminiscences of Dr. S. Weir Mitchell[2]. Walter G. Elmer, M.D. (Read by title.)

15 April 1958

Science and Sentiment: A Study of Oliver Wendell Holmes[1]. Eleanor M. Tilton*,

* By invitation.

[1] Published in *Transactions & Studies*, August, 1958.

[2] Published in *Transactions & Studies*, June, 1958.

Ph.D. (Kate Hurd Mead Class of 1888 Lecture in Medical History, Woman's Medical College of Pennsylvania.)

SECTION ON OPHTHALMOLOGY[3]

20 February 1958

Presentation of Interesting Ophthalmological Cases. Georgiana Dvorak-Theobald*, M.D.

20 March 1958

(Joint Meeting with the Eye Section, Philadelphia County Medical Society)

Functional Results with Contact Lens in Unilateral Congenital Cataracts and High Myopia. Philip G. Spaeth*, M.D.

Retained Glass Foreign Bodies in the Anterior Chamber. P. Robb McDonald, M.D., and Mila J. Ashodian*, M.D.

Retinal Folds in Orbital Expanding Lesions. Thomas R. Hedges, Jr., M.D., and Irving H. Leopold, M.D.

17 April 1958

Electrophysiologic Insight in Ocular Motility. Goodwin M. Breinin*, M.D.

SECTION ON OTOLARYNGOLOGY[4]

19 March 1958

(Joint Meeting with the Section on Otolaryngology of the New York Academy of Medicine at the College of Physicians of Philadelphia)

Stapes Mobilization Operation—Analysis of Technics and Results. Edward H. Campbell, M.D. (Discusser: Greydon G. Boyd, M.D.)

Experiences in Surgical Management of Chronic Mastoiditis with Cholesteatoma. David Myers, M.D. (Discusser: Harry Rosenwasser, M.D.)

[3] Abstracts of presentations at this Section customarily appear in the *American Journal of Ophthalmology* and the *A.M.A. Archives of Ophthalmology.*

[4] Abstracts of presentations at this Section customarily appear in the *A.M.A. Archives of Otolaryngology.*

Unusual Nasal Tumors. Frederick Harbert, M.D., and Stephen Balshi*, M.D. (Discusser: Daniel C. Baker, Jr., M.D.)

Syphilis of the Larynx (Motion Picture). William A. Lell, M.D.

SECTION ON PUBLIC HEALTH, PREVENTIVE AND INDUSTRIAL MEDICINE

21 January 1958

Perinatal Mortality and Preventive Aspects of Prematurity.[1] Norman Kendall*, M.D., G. Gerald Rice*, M.D., M.P.H., and Elizabeth Kirk Rose, M.D.

18 March 1958

Radioactive Fall-Out and Radiation Exposure Hazards.[1] Charles L. Dunham*, M.D., Robert Robbins, M.D., and Chris J. D. Zarafonetis, M.D.

2 April 1958

(Joint Meeting with Pennsylvania Public Health Association, Region One) (James M. Anders Lecture XXXVII)

Health and Hospitals for an Aging Population.[1] Leroy E. Burney*, M.D., M.P.H.

ABSTRACTS

Abstract 1

Fatal Typhoid Fever in an Illegal Immigrant: Case Report. Carmen T. Bello, M.D., and Fred B. Rogers, M.D.

A case of typhoid fever is reported in a Sicilian man who entered Philadelphia as an illegal immigrant from Italy. Violation of civil and quarantine laws in this case resulted in problems of diagnosis, a health threat to attending persons, and death of the patient. Federal investigation instigated by this fatality uncovered an alien-smuggling ring of considerable size, and led to the conviction and imprisonment of several accomplices.

V. R., a 21 year old white male, was admitted to Temple University Hospital in critical condition on March 1, 1957. He had arrived by ship at Philadelphia three days

earlier, without passport or visa, having been sick for two weeks at sea. His illness was characterized by fever, chills, headache, drowsiness and diarrhea. On the day before hospitalization, he passed a bloody stool and became stuporous. He was smuggled ashore by two crewmen and left with relatives. A local physician was called and promptly referred him to the hospital. His past health had been good; he had worked as a farmer in Sicily.

Physical examination on admission revealed a moribund young man. High fever (103°F.), weak pulse and pale skin were evident. There was no skin rash or meningismus. Blood pressure was 85/50. Heart sounds were distant, but breath sounds normal. The abdomen was scaphoid in contour, peristalsis much diminished, and the spleen barely palpable. The presenting picture was one of generalized infection and massive intestinal hemorrhage with shock.

Laboratory study showed hypochromic anemia (hemoglobin 8.6 gms.), and leukopenia (W.B.C. 2500) with lymphocytosis (47%). Urine was scanty and dark in color. Blood smears (thick and thin) were negative for malaria plasmodia. Stool was dark red, semiliquid, and negative for ova and parasites. Blood, urine and stool cultures were negative for *S. typhosa*. Blood agglutinins were: typhoid "O"—positive in 1–80 dilution; "H"—positive in in 1–400; paratyphoid A and B negative (no history of past immunization). Agglutination reactions were negative for Brucella, Proteus OX-19, and Vi antibody. X-ray film of the abdomen detected gaseous distension of the large intestine, extending from the cecum to the sigmoid colon.

The patient's hospital course was steadily downward, with the exception of an abrupt drop of fever (103.4°–100.2°F.) and clearing of mental confusion upon administration of chloramphenicol shortly after hospitalization. Despite vigorous supportive and replacement measures, including 16 gms. chloramphenicol given intravenously and four liters' blood transfusion, the patient experienced more melena and became progressively worse. Muttering delirium appeared and high spiking fever persisted until death on March 5, 1957.

Postmortem examination (Dr. Harvey F. Watts) showed diffuse granulomatous inflammation with necrosis in the ileum, and lymphoid and reticuloendothelial hyperplasia in the mesenteric lymph nodes and spleen. Although ileal ulceration and copious hemorrhage were present, no definite intestinal perforation was found. A clinical and histopathological diagnosis of typhoid fever was made; tissue stains for tuberculosis and fungus infections were negative.

The Medical Examiner of Philadelphia investigated this suspicious death. U. S. Immigration and Public Health Service Officials detained the ship (*S. S. Elga*—Panamanian registry) for a complete search. Several crew members were subsequently held for Federal Court hearings. A large alien-smuggling ring from Italy to the U.S.A. was exposed and interrupted through this key case-fatality.

Abstract 2

THE NATURAL COURSE OF INTERMITTENT CLAUDICATION AS DETERMINED BY CLAUDICATION TESTS. Raymond Penneys, M.D., and Orville Horwitz, M.D.

Repeated claudication tests were carried out on 61 patients having arteriosclerosis obliterans of the lower extremities with intermittent claudication to determine the changes in the patients' capacity to walk when receiving no specific therapy for their claudication. The patients were allowed to walk on a horizontal treadmill, or on a level tile floor ("walking test"), until compelled to stop, or until a ten minute time limit was reached.

The results varied considerably among 61 patients. In 148 of the 424 tests (357 on the treadmill, average speed 141 feet/minute, range 85 to 271 feet/minute; 67 "walking" tests, average speed 241 feet/minute, range 140 to 350 feet/minute) the patient could walk the full ten minute test period. Claudication compelled the patient to stop

before the ten minute time limit was reached in only 201 tests. In 26 tests, walking was terminated because of the onset of angina pectoris or severe dyspnea, and in 49 tests it was terminated for miscellaneous reasons (symptoms in the extremities other than claudication, technical failure, generalized tiredness, poor coordination on the treadmill).

The claudication response of the same patient even on the same day at the same speed may be different. In 7 of 73 instances where two tests were done on the same day claudication appeared in a different extremity in the two tests.

There was no consistent change in the claudication time in 29 patients who had repeated tests at the same speed over an average period of 11 months (3 to 18 months). In 16 patients the time was longer on their most recent test than on their first test, in 8 it was shorter, and in 5 the time remained the same. The ages of the patients and the number of years they had their claudication were about the same for those whose claudication time became longer as for those whose time became shorter.

Fourteen of these 29 patients could, at one time or another, walk the full ten minute time limit, apparently "cured" of their claudication as determined by the claudication tests.

These aspects of the natural course of intermittent claudication must be appreciated in drawing conclusions as to the efficacy of any specific therapy.

Abstract 3

A New Therapeutic Agent for Acute Nickel Poisoning. F. William Sunderman, M.D., Ph.D., and F. William Sunderman, Jr., M.D.

The therapeutic effectiveness of the dithiocarbamates has been studied in experimental animals receiving lethal inhalations of nickel carbonyl. Of the various derivatives investigated the sodium salt of diethyldithiocarbamic acid (Dithiocarb) proved to be the most effective and least toxic.

Clinical observations and laboratory studies were made on eleven persons who received Dithiocarb following acute exposure to the vapors of nickel carbonyl. Symptoms of nickel carbonyl poisoning in all of the exposed individuals were quickly relieved after the ingestion of Dithiocarb. Delayed reactions and the periods of convalescence were brief.

Following the administration of Dithiocarb, the concentration of nickel in the urine became increased in each of the cases of severe exposure.

Abstract 4

Factors Contributing to Mortality in Experimental Hemorrhagic Pancreatitis. Alexander W. Ulin, M.D., and Irvin H. Sokolic, M.D.

Using the method of Archibald, bile injection pancreatitis was produced in 6 dogs. This experimental pancreatitis was uniformly fatal within seventy-two hours. The serum amylase values ranged from 12,000 to 32,000 Somogyi units (normal range for dogs, up to 2,000 units). Gross, microscopic and bacteriologic studies were recorded. The following factors were then studied as causes of mortality: (1) circulating enzyme; (2) circulating toxin from necrotic pancreas; (3) peritonitis; (4) blood volume.

Using dl-ethionine, a metabolic inhibitor that interferes with enzyme production by acinar cells of the pancreas, 27 dogs were prepared using various dosage schedules and then pancreatitis was produced by the standard technique. Two animals survived and 25 died within three to five days. These animals did not develop marked rises in serum amylase and lipase. Pathological study revealed hemorrhagic pancreatitis, but with significantly less hemorrhage and necrosis. Thus, while the course of the pancreatitis was quantitatively altered to some extent, the suppression of circulating pancreatic enzymes did not prevent a fatal outcome. This confirmed the work of Popper and Ellison who used soy bean inhibitor to counteract the effect of circulating trypsin in similar

experiments. They found that neutralization of this circulating enzyme did not prevent mortality.

The release of a non-specific toxin from the necrotic pancreatic tissue was then investigated. Acute pancreatitis was produced in a donor dog. The entire pancreas was then excised and homogenized in a Waring blender. To this was added 100,000 units of penicillin and one-half gram of streptomycin. The entire mash was then injected into the peritoneal cavity of a second animal through a trochar incision. This experiment was repeated three times on three different recipient animals. Serum amylase values ranged from 5,000 to 9,000 units. However, the animals remained perfectly well and alert and clinically gave no indication of morbidity.

The problem of peritonitis and infection was explored in 3 animals. The use of antibiotic therapy prevented mortality in each instance. This confirmed the work of Fine and his group.

Finally, we investigated the systemic effect of the experimental pancreatitis in the animal when the pancreas was transplanted in an extraperitoneal position. This was accomplished in two stages. In the first stage the dog's pancreas was bisected and the portion including the main duct and part of the duodenum was transplanted extraperitoneally in a subcutaneous position with blood and nerve supply intact. In some animals the proximal pancreas was excised and in others was left intact. (In 4 animals an attempt to transplant the entire pancreas was uniformly fatal, apparently, because of difficulty with blood supply to the explanted pancreas.) In this preparation six controls were used to produce bile injection pancreatitis in the intraperitoneal proximal half of the pancreas. Five of these animals died with the usual chemical, pathological and bacteriological findings. The extraperitoneal pancreas did not show evidence of significant pancreatitis. When the same type of pancreatitis was produced in 11 extraperitoneal preparations, 9 survived and 2 died. The same experiment was then repeated with 8 dogs prepared with the extraperitoneal and intraperitoneal halves of pancreas. In four, pancreatitis produced in the intraperitoneal portion of the pancreas produced 100% mortality. In the experiment on the 4 extraperitoneal injected ducts all the animals survived. Blood volumes were measured by the R.I.S.A. technique. The data indicated a marked reduction in blood volume for the intraperitoneal cases of pancreatitis. For the pancreatitis in the extraperitoneal position, there was little change in the measured blood volumes. Furthermore, it was noted by gross and microscopic study that in the animals who survived there was no inflammatory change in the intraperitoneal portion of the pancreas.

We concluded from these experiments that bacterial peritonitis and hypovolemic shock were significant factors contributing to mortality in experimental bile injection pancreatitis.

Abstract 5

Adrenal Cortical Carcinoma Causing Feminization in an Adult Male: Hormonal Considerations and Heterotransplantation of the Tumor in Guinea Pigs. Lewis C. Mills, M.D.

Adrenal cortical carcinomas associated with excessive estrogen production leading to feminization in the male are quite rare, as only 34 cases have been reported in the medical literature. Since only a limited number of steroid assays were done in any of these patients, it seemed desirable to report the clinical and laboratory findings in another patient in whom excretion of 17-hydroxycorticoids, 17-ketosteroids, estrogens and pregnanediol were measured before and after surgery. Associated with the clinical manifestations of feminization, excretion of all of the above steroids was increased. During ACTH stimulation, there was no rise in urinary 17-ketosteroids, and only a subnormal rise in 17-hydroxycorticoids.

At surgery an adrenal carcinoma weighing 1750 grams was removed. Postoperatively

the patient's libido increased and the gynecomastia subsided. Excretion of the above steroids decreased following surgery, and, with the exception of pregnanediol, were within normal limits.

The tumor of this patient was successfully transplanted into the anterior ocular chamber of the eye of guinea pigs. Transplants of the tumor maintained their ability to secrete estrogens.

Abstract 6

PRIMARY PULMONARY HYPERTENSION IN XANTHOMATOUS BILARY CIRRHOSIS. Edward S. McCabe, M.D.

The frequency with which the disease primary pulmonary hypertension occurs is very difficult to assess from the literature. Moschcowitz reported varying degrees of pulmonary arteriosclerosis in 6.5 per cent of 770 consecutive necropsies, while Brenner showed some pulmonary vascular sclerosis in 23 of 31 routine autopsies, but with right ventricular hypertrophy existing in one alone, 3.3%. It would seem that the characteristic lesion must occur in the lungs proximal to the capillaries and that xanthomatous biliary cirrhosis will predispose to pulmonary hypertension in a fair number of cases.

The case to be presented has been published earlier in *Gastroenterology*, March 1952, but this additional material has been gathered over a two-year follow-up period. The diagnostic features are:

1. Sex-linked characteristic (female only).
2. Skin xanthoma of the plain and tuberous variety.
3. Hepatomegaly and occasionally splenomegaly.
4. Mild obstructive type of jaundice of years duration.
5. Extremely high values for total cholesterol as well as phospholipid.
6. Transparent serum showing a low value for neutral fat.
7. Pericholangiolithic biliary cirrhosis.
8. Reddish maculopapular eruption on extensor surfaces.
9. Primary pulmonary hypertension.

The patient was readmitted to the Philadelphia General Hospital during 1952–3 with chief complaints of swelling of the abdomen and the lower extremities, and in addition some bleeding per rectum on bowel movements. A serum lipid pattern showed a striking reduction from the original:

1953	*mg./100 cc.*	*1949*
288	Cholesterol	1225
100	Cholesterol esters	138
35	Cholesterol per cent	11.5
283	Phospholipid	2285
300	Total fatty acids	1160
46	Neutral fat	298
684	Total lipids	2900

This reduction was accomplished by abstaining from alcohol and keeping to a low fat diet, initially a Rice diet, with negative or isocaloric balance later increased in protein content. Vitamins and iron were given as supplements while on the Rice diet.

The buccal mucosal lesions have gone and the palmar lesions have almost disappeared. The xanthelasma is less marked.

Certain allied metabolism defects were a flat glucose tolerance curve due to increased utilization rather than impaired absorption, probably enhancing lipogenesis. It appears that a condition of adrenal unresponsiveness exists and that phospholipid turnover is accelerated. Galactose seemed to be easily absorbed but not utilized since clearance was impaired. A fat tolerance test (Schwartz units) gave a flat curve proving that phospholipid formation more than kept pace with demand.

The heart showed a systolic mitral murmur with a split second sound at the pulmonic area on the phonocardiogram. The left auricle was not enlarged. The electrocardiogram tracing showed marked right ventricular hypertrophy and vector analysis indicated an abnormally widened QRS-T angle. Cardiac catheterization studies in 1954 revealed pressures in the right atrium 8/3, right ventricle 85/7, pulmonary artery 95/40, with a capillary wedge pressure of 15. Oxygen levels

were within normal limits, and there was no evidence of a left to right shunt.

This patient died early the following year in the Temple University Hospital Accident Ward, presumably from a ruptured esophageal varix. Due to staff changes in the Philadelphia Coroner's Office an autopsy report was not obtained. Clinically a portal cirrhosis coexisted terminally. From a rapid survey of the literature and from personal knowledge of catheterization studies in one other case it would seem that primary pulmonary hypertension is a feature of this disease.

Abstract 7

TRANSPLANTATION OF HOMOLOGOUS THYROID AND PARATHYROID GLANDS: CASE REPORTS. Julian A. Sterling, M.D.

Three patients who received homologous transplants of thyroid and parathyroid glands, utilizing anastomoses between the host and the donor's vascular pedicles, no longer have signs or symptoms of hypoparathyroid tetany. In addition, one patient is free of pre-existing hypothyroidism. The other two patients, although not taking thyroid extract and being reasonably free from symptoms of hypothyroidism, do not have evidence of thyroid cell activity in the transplant at this time.

Presumptive clinical evidence indicated that parathyroid cell function is present in these three patients, due to persistence of homologous gland tissue which was transplanted 2, 3, and 5½ years previously.

Abstract 8

CONGENITAL VIRILIZING ADRENAL HYPERPLASIA IN IDENTICAL TWINS. Norman G. Schneeberg, M.D.*, Arthur Steinberg, M.D.*, Maurice Malen, M.D.*, and Benjamin Chernoff, M.D.*

The occurrence of congenital virilizing adrenal hyperplasia in more than one member of a family is not unusual and suggests a genetic cause. Its existence in identical twins, however, is extremely rare and has only been well authenticated in a single instance (1). This report details the findings in twin female pseudohermaphrodites age 7½ years. They showed enlarged clitori at birth; in the ensuing years mild hirsutism, acne, accelerated somatic growth, precocious dental maturation, deepening of the voice, the appearance of axillary and pubic hair, and accelerated epiphyseal maturation occurred. At age 7½ years they were 58 inches tall, weighed 80–83 pounds, showed adult dentition and a bone age, by x-ray, of 16–19 years. The clitori were markedly enlarged and the vaginal introitus and urethra were separate and in normal relationships. A tiny cervix could be visualized after vaginal dilatation. Urinary 17-ketosteroids were 10 to 48 mg. per 24 hrs., corticoids were slightly low, and pregnanetriol was present in large amounts (70–74 mg./24 hrs.). There was no hypertension or salt-losing tendency. The sella turcicae were normal; intravenous urography and retroperitoneal air insufflation did not show an adrenal tumor. Histologic study of skin biopsies revealed a female sex chromatin pattern. Blood grouping studies indicated a 98% probability that they were identical twins. The urinary 17-ketosteroids were markedly reduced during an intravenous infusion of hydrocortisone and elevated three to fourfold after intravenous ACTH.

Therapy with 100 mg. of cortisone given intramuscularly daily did not produce a satisfactory reduction of the urinary 17-ketosteroids but there was subsidence of acne, and breast budding, slight elevation of voice pitch and loss of hirsutism was noted. A saline suspension of "long-acting" hydrocortisone was given by weekly intramuscular injections. The urinary 17-ketosteroids were suppressed satisfactorily but there were Cushingoid side effects. Oral prednisone similarly induced Cushingoid changes without successful adrenal suppression. A second trial of intramuscular cortisone in doses of 50 down to 25 mg. per day intramuscularly reduced the 17-ketosteroids to an average excretion of 13 mg. per day. Nine-alpha-fluorohydrocortisone was given orally in doses of 5 mg. per day but had to be stopped because of edema and marked

hypertension. Successful therapy was finally achieved with 25 mg. of cortisone in one twin and 8 mg. of prednisone in the other given intramuscularly every other day. Amelioration of virilization, and full pubertal breast development was attained. Menarche has not appeared.

Congenitally hyperplastic adrenal cortices secrete virilizing adrenal steroids during prenatal life and, in the female, distort the normal differentiation of the genital tract towards a male pattern. Most cases exhibit an enlarged clitoris and a persistent urogenital sinus into which the uterine body and urethra empty. Occasionally, as in the twins reported here, the disturbance develops later in intrauterine life and the urethra and vagina are separate. This disease is viewed as an inborn deficiency of the adrenal cortical enzymes responsible for the conversion of 17-hydroxyprogesterone to hydrocortisone (2). The synthetic pathway is deviated from hydrocortisone production towards the elaboration of pregnanetriol and various androgenic 17-ketosteroids. The deficiency of hydrocortisone permits adrenocorticotrophic hormone (ACTH) to act unimpeded upon the adrenal cortex to further stimulate adrogen production. The administration of cortisone inhibits ACTH, and interrupts the vicious cycle of androgen synthesis.

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2. Jailer, J. W., Virilism, Bull. N. Y. Acad. Med. **29:** 377, 1953.

Abstract 9

Report of a Case of Nocardiosis Due to Nocardia Asteroids Involving the Lungs. Frans J. Barends, M.D.

Nocardiosis is a sub-acute or chronic infectious disease of granulomatous or suppurative nature, caused by an aerobic, gram-positive, inconstantly acid-fast species of the family *Actinomycetaceae*. The organism most frequently isolated is *Nocardia asteroides*. Nocardiosis is recognized as a separate and distinct disease and therefore should not be confused with actinomycosis.

The medical literature has been reviewed and a new authentic case has been added to the 58 previously reported. This patient was a 47 year old male who presented a history of cough, chest pain, weakness, anorexia, weight loss, night sweats, chills and fever for the period of one month after which he was admitted to the Harrisburg Polyclinic Hospital, Harrisburg, Pennsylvania, on February 14, 1956. Physical findings at that time included fever, rapid pulse and respirations, inflamed pharynx covered with dry exudate having a foul odor, and dullness to percussion over the lower portions of both lung fields.

Chest x-ray films showed extensive infiltration throughout the lower halves of both the upper lobes; this was thought to be due to an acute inflammatory process and the possibility of tuberculosis was suggested.

Laboratory tests were not remarkable except for a high leukocyte count. Skin tests were negative in a complete P.P.D. series; sputum cultures and smears were negative for mycobacteria, as was guinea pig inoculation. The patient was placed on penicillin, chloromycetin, liquids and supportive therapy.

Because of the continuing suspicion of tuberculosis, streptomycin and isoniazid were administered later on. The patient expectorated a great deal of thick, purulent sputum, became progressively worse and expired on the 12th hospital day.

Autopsy showed almost complete consolidation of the lower portions of both upper lobes; gross sections showed large zones of ulceration and cavitation. Microscopic examination of both lungs revealed extensive consolidation and involvement of the alveoli, primarily with an acute inflammatory exudate. Tissue microscopic sections of the lung revealed many slender, gram-positive, branching hyphae invading the tissue.

Antemortem cultures of sputum, as well as cultures obtained from the lung at autopsy, showed the growth of a fungus, later identified by the Communicable Disease Center Labora-

tory, United States Public Health Service, at Chamblee, Georgia, as *Nocardia Asteroides* (pathogenic for guinea pig).

The epidemiology of nocardiosis has already been reported. Differential diagnosis from tuberculosis, actinomycosis and from saprophytic mycobacteria is often difficult. Nocardia infections have the tendency to disseminate from a primary nocardial infection in the lungs (the respiratory tract is the usual portal of entry). Almost any organ may become involved, but subcutaneous and central nervous tissues are the most common secondary sites. The clinical aspects have also been reviewed.

The radiographic findings in pulmonary nocardiosis vary greatly. A dense area of lobular or lobar consolidation will often be seen. This area of consolidation may contain radiolucent areas (suggesting lung abscess), or cavitation may occur (suggesting tuberculosis).

Until 1944 all cases of Nocardiosis terminated fatally. The treatment employed in this case has been noted. Sulfadiazine is the drug of choice. A combination of sulfadiazine with streptomycin or other broad spectrum antibiotics should be evaluated according to the sensitivity tests. Penicillin therapy is useless. Surgical drainage should be employed when indicated.

Abstract 10

Hemoptysis in Older Men. Katharine R. Boucot, M.D., David A. Cooper, M.D., and William Weiss, M.D.

Hemoptysis is alarming to both patient and physician, especially in older men in whom lung cancer must be considered. The relationship between hemoptysis and lung cancer is a major interest of the Philadelphia Pulmonary Neoplasm Research Project,* a study begun in December, 1951, in an effort to increase the percentage of survey-detected "curable" lung cancer by a more intensive study of older men with respiratory symptoms and normal chest X-rays. In addition, the Project is studying the chronologic relationship between symptoms and radiologic abnormalities to delineate the characteristics of curable cancer and illuminate the natural history of this grave disease.

Between December 4, 1951 and December 3, 1955, 6137 men over age 45 joined the Project. The design of the study calls for semiannual chest X-rays and interviews by a lay staff member over a 10-year period.

Thus far, hemoptysis has been the most important symptom in relation to lung cancer, with an over-all prevalence of five per cent for the entire group. Those men who were referred to the Project for pre-employment examinations had an hemoptysis prevalence of two per cent and those with negative films a prevalence of three per cent—suggesting that the hemoptysis rate of two to three per cent may be a minimal figure for men over age 45.

Only six of the 32 men with a diagnosis of lung cancer were alive one to five years after their hemoptyses, strengthening the impression that hemoptysis appears late in the course of untreated bronchial cancer.

Hemoptysis in older men is as important as it appears to be to physicians and lay persons since one out of every two men over age 45 who had this symptom had an adequate diagnosis established.

* Supported by grants-in-aid of research from the American Cancer Society and its Philadelphia Division, the Philadelphia Tuberculosis and Health Association, and a private foundation.

Abstract 11

A Case of Generalized Cryptococcosis Treated with Amphotericin B. John G. Rogers, M.D., and Perry S. MacNeal, M.D.

Cryptococcosis has a uniformly poor prognosis. Rare cases of spontaneous recovery or recovery following treatment with sulfonamides has been reported. A case of meningitis treated with Amphotericin B was reported in 1957. The case presented is that of a 55 year old white female who had suffered pain in the buttock and left thigh for two months. One month prior to admission she noted a gradually increasing soft tissue mass in the left anterior chest, which later was sponta-

neously drained. There was no associated fever, weight loss or malaise. Physical examination on admission revealed blood pressure 160/90, temperature 98, pulse 100, respirations 18. Positive findings included good nutrition, a soft tissue mass over the left 6th rib and 7th rib in the mid-axillary line which contained a draining sinus, tenderness over the left ischial tuberosity and pain on motion of the left hip. Routine urinalysis and complete blood count were normal. The corrected sedimentation rate was 15 mm./hr. The drainage was negative for acid fast bacilli.

X-ray examinations revealed an area of bone destruction measuring 3 cm. in diameter in the left iliac body. There was also increased density of the left ileo-psoas shadow. Chest x-ray revealed a soft tissue mass in the left axilla, as well as partial destruction of the 6th rib and underlying localized pleural thickening. The lung fields were clear.

Skin tests with histoplasmin and blastomycin were negative. Tissue and cultures from the subcutaneous abscess in the chest wall were examined by Dr. Norman F. Conant of Duke University Medical School. He reported the organism as representing *cryptococcus neoformans* on tissue stains. The organism was not cultured.

The patient was treated with intravenous amphotericin B from 1/11/57 to 2/2/57. The dosage ranged from 20–50 mgm. intravenously daily given in a drip of 1,000 cc of 5% glucose in water. The patient reacted with headache, nausea, anorexia, vomiting, fever as high as 103°F. and chills. The dose had to be occasionally decreased or omitted because of the severe side effects. Acetylsalicylic acid, codeine, pyribenzamine, Gelusil, milk and cream were all used for symptomatic treatment of the side effects.

On 1/24/57 the chest lesion was smaller and there was less pain on walking. On 1/26/57 the abscess drainage had ceased.

On 1/29/57 an upper gastrointestinal x-ray was normal. Laboratory studies, while under treatment, revealed BUN 10, total bilirubin 0.6. Hemoglobin fell to 9.5 grams and the hematocrit to 32. Total white count remained normal. A PSP excretion test revealed 65% of the dye excreted in 20 minutes.

The patient has remained clinically well to date. Repeat x-ray examination on 9/3/57 showed excellent healing of the left rib and left iliac wing with no soft tissue abnormalities.

It therefore seems that a remission has been induced in a case of generalized cryptococcosis with the intravenous administration of amphotericin B over a three week period. Side effects were unpleasant but did not interfere with completion of treatment.

Abstract 12

PULMONARY EMBOLISM AS A CAUSE OF PLEURISY WITH EFFUSION. Albert L. Sheffer, M.D., Dick D. Harrell, M.D., and Harold L. Israel, M.D.

Pulmonary embolism and infarction are frequently accompanied by pleural effusions, but these are usually small and overshadowed by other manifestations of the embolism. It is not generally appreciated that pulmonary embolism can be responsible for clinical, radiologic and laboratory changes which stimulate in many respects tuberculous or neoplastic effusions.

Between August 1955 and January 1957, a diagnosis of pulmonary embolism was made in 90 patients at the Graduate Hospital of the University of Pennsylvania. In 33 instances (45%) there was radiologic evidence of pleural effusion. In the majority of instances the clinical picture was not of pleurisy with effusion, and for this reason thoracentesis was performed in only two patients. These two cases and five subsequent cases in which pulmonary embolism produced pleural effusions which lead to serious concern about tuberculosis or neoplasm are described briefly.

Recent experience has challenged commonly held beliefs regarding pleurisy with effusion. It has long been considered sound clinical practice to treat as tuberculous all pleural effusions despite the inability to demonstrate tubercle bacilli. Justification for this

policy was provided by the frequency with which tuberculosis subsequently developed in patients with idiopathic effusions. Similarly, it has been generally thought that large bloody pleural effusions are almost invariably due to malignant lesions involving the pleura. Transient effusions which are not attributable to neoplasms or tuberculosis are often blamed on pneumonia or viral infection. Effusions are well recognized complications of bacterial pneumonias, but relatively uncommon since the advent of antibiotics. Viral infections are rarely the cause of pleural effusion.

As a result of the development of pleural biopsy techniques, presumptive diagnoses of tuberculosis are no longer justified. In cases of pleurisy in which aspiration or surgical biopsy of the parietal pleura fail to demonstrate tuberculosis or malignancy, serious consideration must be given to pulmonary embolism.

College Notes

WILLIAM HARVEY PERKINS AWARD IN PREVENTIVE MEDICINE AND PUBLIC HEALTH

The Section on Public Health, Preventive and Industrial Medicine of the College of Physicians of Philadelphia announces the establishment of an annual William Harvey Perkins Award of $100 for the best essay on a subject pertaining to preventive medicine and public health submitted by a student enrolled in any one of the five Philadelphia medical schools. This award has been established by the Section to honor William Harvey Perkins, Fellow of the College and former Dean and Professor of Preventive Medicine at the Jefferson Medical College. Dr. Perkins, whose book, *Cause and Prevention of Disease* (1938), was a significant contribution to the science of preventive medicine, was formerly Professor and Director of the Department of Preventive Medicine and Director of the Hutchinson Memorial Clinic at the Tulane University of Louisiana, New Orleans, and Acting Professor of Medicine, Chulalangkarana University, Bangkok, Thailand. In a College Anders Lecture on 'The Value of Health Centers to the Community' (1951), Dr. Perkins forecast community health centers such as the ten units now an integral part of the Philadelphia Department of Health.

The subject of papers submitted for the William Harvey Perkins Award may be any aspect of preventive medicine or public health. All essays must be received by the College of Physicians on or before April 1, 1959. The manuscripts must be typewritten, on one side of a page only, with double spacing and wide margins. Students who are interested are invited to utilize the resources of the Library of the College of Physicians of Philadelphia, or any other reference material desired. John J. Hanlon, M.D., Chairman of the Section, or Fred B. Rogers, M.D., Clerk, will assist any student so desiring, in choosing a topic for study, in reviewing material or in preparing the manuscript. The annual prize essay, if deemed suitable, will be published in the College's *Transactions & Studies*.

WILLIAM OSLER MEMORIAL

The Medical Historical Club of Toronto plans to erect a memorial to Sir William Osler on the site where this great physician was born, at Bond Head, on July 12, 1849. This memorial is to be a cairn, erected on a plot of ground purchased by the Club, marking the site of the church in which Osler's father was Rector at the time of this son's birth. The Club will be pleased to receive contributions from anyone who wishes to assist in the completion of this memorial. Checks should be made payable to the Medical Historical Club and sent to the Secretary, Dr. Eric A. Linell, 253 Blythwood Road, Toronto 12, Ontario, Canada.

Elected a Fellow of the College of Physicians of Philadelphia in January, 1885, Osler served on its Library Committee while in this city and continued his cordial association with the College until his death in 1919. The Section on Medical History has accordingly sent a donation to the Medical Historical Club of Toronto to assist in the erection of this memorial marking the site of Sir William Osler's birth.

NEW FELLOWS

The following were elected Fellows on the dates listed:

8 January 1958

Alfred S. Avella, Jr.
Samuel Baer
Simon Ball
Edward W. Closson
Milton J. Freiwald
Toby A. Greco
Samuel Levine

Edward B. LeWinn
David Y. P. Lin
Arthur Lipschutz
Ralph W. Mays
Samuel R. Moore, Jr.
Rowan C. Pearce, Jr.
Lillian Rachlin
Albert H. Shafer
Cyril L. Velkoff

5 February 1958

John J. Hanlon
Jacob Katz
Coleman W. Kovach
Truman G. Schnabel, Jr.
Isaac Sharpless
Joseph A. Splendido

5 March 1958

Frank P. Brooks
George W. Deitz, 3rd
George D. Geckeler
Wallace Gilfillan McCune
Leonard S. Girsh
Charles K. Kirby
Thomas A. Loftus
Alma Dea Morani

14 May 1958

John Deaver Alexander
John L. McClenahan
David J. Reinhardt, III
Hammell Pierce Shipps
Irving Woldow

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Immunology of Clinical Allergy[1]

By OSCAR SWINEFORD, JR., M.D.[2]

I AM grateful for the prestige which your invitation to deliver the 1958 Hira S. Chouké lecture has conferred upon me. I am particularly pleased with this opportunity to address the members and guests of the College of Physicians of Philadelphia which is so rich in the best of medical traditions.

So far as I know this is the only lectureship of its kind which emphasizes allergy. It is to be hoped that the combined efforts of the Allergy Foundation of America and the National Institute of Allergy and Infectious Diseases, plus the efforts of perceptive individuals like Dr. Chouké, will lead soon to the proper emphasis of allergy in our medical schools. Allergic diseases stand third in the National Institutes of Health list of causes of illness. It is high time our Curriculum Committees faced up to the fact that graduates of medical schools should be, but are not, as competent in allergy as they are in cardiology, hematology, gastroenterology and endocrinology.

The Immunology of Clinical Allergy is a broad subject, too broad for detailed analysis here. By way of compromise, certain aspects of the subject have been selected in the hope that the discussion will stimulate the neophyte in allergy to pursue its many fascinating facets. A selected reading list is appended. Considerable emphasis will be placed upon the potential roles of non-allergic mechanisms in some of the syndromes which have long been considered the preserves of the allergist. Diagrams will be used freely to summarize the conventional portions of the discussions of anaphylaxis, desensitization, the atopic disorders, drug and serum reactions, and contact dermatitis. The immunology of the tuberculin, Arthus, collagen disorders and id reactions will not be discussed. It will soon become evident that there are important gaps in our knowledge of these phenomena, gaps that stand as provocative challenges to the eager young investigator.

All allergic reactions, regardless of type, are thought to be mediated by the interaction of antigens, antibodies and tissues. These are spoken of as the allergic triad. Tables I and II summarize some of the factors involved in the interaction of this triad.

The antigens which sensitize and elicit anaphylactic and atopic reactions are proteins or combinations of haptens with proteins. The usual sources are foods, pollens, molds, organic dusts, animals, and drugs. The antigens in contact dermatitis will be discussed later.

In spite of the fact that antigens are the key factors in allergic reactions and that important clinical roles have been assigned to hundreds

[1] Hira S. Chouké Lecture II, College of Physicians of Philadelphia, 5 March 1958.

[2] Professor of Internal Medicine, Allergy-Arthritis Division, University of Virginia School of Medicine, Charlottesville, Va.

TABLE I

COMPONENTS OF ALLERGIC REACTIONS

Allergic Triad			
	Antigens	of infinite variety	
		tissue	*type of allergy*
	Tissues	Mast Cells Endothelium Smooth Muscle	Anaphylaxis and Atopy
		Epidermis	Contact
		Many Cells	Tuberculin
		Antibody	*Mediates*
	Anti-bodies	Precipitin	Anaphylaxis Serum Reactions Passive Desensitization
		Reagin	Sensitization-Human
		Blocking	Protection-Human
		Comp. Fixing	Most Reactions

(Antibodies which mediate delayed allergic reactions have not been characterized satisfactorily)

TABLE II

MECHANISM OF ALLERGIC REACTIONS

2 PHASES

A. Sensitization
- Active
  - Injected antigen → antibody
  - Tissue Cells fix antibody
- Passive
  - Injection of preformed antibody
  - Tissue cells fix antibody

B. Reaction

Antibody + Tissues + Antigen → -H- Substances → Asthma, Hay fever, Urticaria, Angioedema, Atopic dermatitis, Anaphylaxis, Serum Reactions

of them, they have received surprisingly little attention from the immuno-chemists. Some ineffectual efforts have been made to identify the active principles of house dust and ragweed pollen. Little effort has been made to identify the immunologically active constituents of other allergens, although the ground work for this was laid years ago by Landsteiner and by Heidelberger and their associates in their studies of haptens and bacterial antigens.

The role of the tissues has not been a popular subject for the investigator, beyond the rather extensive studies of the histological sequences in the several types of allergic reactions. It is not known, for example, how nor why the interaction of antigens and antibodies in vivo should set off the chain of events which results in anaphylaxis or asthma. A brief composite of several hypotheses is that the interaction of antigen-antibody and complement activates pro-enzymes which damage mast and other cells with consequent release of a group of anaphylactically active materials referred to as the H-substances. The response to these irritants is the microscopic and gross changes which are recognized as allergic reactions.

The role of antibodies will be discussed later.

Anaphylaxis provides the closest experimental immunological analogue to the common clinical allergic syndromes such as asthma, hay fever, urticaria, angioneurotic edema and serum reactions. Much of the current knowledge of atopic mechanisms stems from studies of anaphylaxis. Anaphylaxis and other allergic manifestations should be thought of as occurring in two phases (Table II). Sensitization, the first phase, may be brought about actively or passively. Sensitization is symptomless. The animal seems to be perfectly normal until antigen is introduced. Serum from an actively sensitized animal may, however, sensitize a normal animal passively. The reaction, the second phase, occurs when antigen is introduced, thus completing the allergic triad of antigen-antibody-tissues, with subsequent release of the H-substances. The term H-substance (histamine-like) was coined to emphasize the role of histamine and to give cognizance to the presence of other active substances. The various components of the H-substance which have been thought to play roles in anaphylaxis and other allergic reactions are listed in Table III. The net effect of the systemic release or activation of these substances is anaphylaxis. The local effect is allergic inflammation which, presumably, has most of the responses to in-

jury that are listed in Table III. Whether some of the materials which have been identified with the H-substance are the cause or the effect of anaphylaxis has not been determined.

Anaphylaxis occurs in all degrees of severity. The milder reactions are more apt to follow subcutaneous injections of antigen. The severe and fatal reactions usually follow intravenous injections or inhalations of aerosols of antigens. The chief shock organ in the guinea pig is the bronchial tree. The chief symptoms are scratching of the mouth and nose, roughening of the hair, cough and expiratory dyspnea. The shocked animal either gets better or worse in a few minutes. It may recover from any degree of expiratory dyspnea, even convulsive gasps. It usually dies promptly if it develops convulsions, however.

The respiratory disturbances are strongly reminiscent of hay fever and asthma.

Species differences in the manifestations of anaphylaxis are well known. The guinea pig is the conventional experimental animal. The rabbit, dog, mouse and rat have been studied less extensively. Anaphylaxis in man, which was common when prophylactic and therapeutic antisera were used extensively, is becoming common again, chiefly from penicillin. Man differs from experimental animals in that he has a variety of shock organs. Each allergy victim has one or more organs which bear the brunt of the interaction of the allergic triad.

Desensitization: Injections of properly selected extracts of airborne antigens provide good results in a high percentage of victims of respiratory allergy. The mechanisms by which injected antigens relieve atopic allergic manifestations are controversial. Some idea of the problems involved can be obtained by comparing the methods by which allergic guinea pigs can be made to tolerate shocking doses of antigen with current methods for treating atopic respiratory syndromes.

Guinea pigs can be desensitized:

(1) Actively, by injecting several sub-reacting doses of antigen subcutaneously or intraperitoneally prior to the intravenous challenging doses of antigen. This can be accomplished in a few hours by 3 to 6 increments of antigen. The small doses of antigen are supposed to saturate fixed antibody (Table IV) at a rate which causes no or imperceptible local reactions. Just what happens when antigen "saturates fixed antibody" has not been determined. Desensitization by this method is temporary, since guinea pigs will become highly sensitive again in about two weeks. The desensitizing doses of antigen are presumed to elicit sensitizing antibodies which resensitize the animals actively, regardless of whether the original sensitization is produced actively or passively.

Effective relief of pollen hay fever and asthma can be provided within a few days by 1 to 3 daily injections of appropriate extracts during the pollen season, so-called co-seasonal treatment. There has been no attempt to ex-

TABLE III

Tissue Response to Injury

(Modified from Feldberg)

H-Substances	Responses by No.	Response
Histamine	2, 8, 9	1. Sticky Capillaries
Serotonin	2, 4, 9	2. + Cap. Permeability
Complement	2, 4, 7, 9	3. Spread of Exudate
Hyaluronidase	2, 3	4. Smooth Muscle Contraction
Polypeptides	1, 2, 5	5. WBC Migration
S-R-S	4, 8	6. Proteolysis
Globulins	1, 2, 5	7. Cell Lysis
Eu & Pseudo	6, 7, 8	8. Dilate Arterioles
		9. Anaphylaxis

TABLE IV

Experimental Desensitization

Hypersensitive animals made refractory by

1. Gradual saturation of fixed antibody by small doses of antigen
2. Injecting excess of circulating antibody to neutralize antigen before it reacts with fixed antibody
3. Exhausting of fixed antibody by sublethal reactions
4. Active production of excess circulating antibody by repeated doses of antigen
5. Non-specific injections of peptone, antiallergic drugs, etc.

TABLE V

EXPERIMENTAL-ANAPHYLACTIC

Sensitize	Protect in Excess
Produced in response to antigen	
Inseparable from precipitin (?)	
CLINICAL—HAY FEVER, ASTHMA, ETC.	
Reagins—Sensitizing Antibodies	
Arise naturally—hard to produce	
Sensitize normal tissues transiently	
Allergen + reagin → immediately positive skin test	
Local desensitization follows reaction with allergen	
Increased early in treatment	
Decreased late in treatment	
Destroyed by 60° C. in one hour	
Separate reagins for separate pollen fractions, i.e. highly specific	
Not demonstrable in all cases	
Increase during season in untreated cases	
Bind very little allergen in vitro	
Passive sensitization is transient, weeks	
Less common to fraction II of ragweed extract than to fraction I	
Do not permeate placenta	
Blocking (Thermostable—Protecting—Neutralizing) Antibodies	
Do not occur naturally	
Readily produced by injecting allergen	
Do not sensitize	
Do not combine with reagin	
Not destroyed by 60° C. in 1 hour, but are in 24 hours	
Combine with allergen to neutralize it	
i.e. reagin + allergen → Reaction	
+ blocking antibody → No Reaction	
i.e. compete successfully for allergen with reagin	
Combination with allergen is transient	
Confer active and passive protection	
Subside when treatment stops	
Return rapidly when treatment is resumed	
Diffuse from injected sites within 24 hours	
Do not delay nor prevent sensitization by reagin	
Highly specific, i.e. separate blocking antibodies for ragweed fractions I and II, but infrequent for fraction II	
Demonstrated for ragweed and grass so far	
Same for dwarf and giant ragweed	
Permeate placenta	

plain the mechanism by which co-seasonal treatment provides relief, except to compare it with this form of active desensitization in the guinea pig. The titer of circulating sensitizing antibody is not affected by co-seasonal therapy.

(2) Excess circulating antibody can be produced by repeated doses of antigen, another form of active desensitization. The circulating antibody will prevent reactions to small doses of antigen, presumably by combining with the antigen before it reaches the fixed antibody. Circulating antibody seems to neutralize antigen more effectively when it is absorbed slowly from the subcutaneous or peritoneal deposits than from intravenous injections. It is difficult to produce enough antibody in the guinea pig to prevent reactions to more than a few multiples of the minimal fatal intravenous anaphylactic dose of antigen. This is the method the clinician uses to prevent allergic reactions to pollens, dusts, mold spores, etc. Allergen injections in man are supposed to elicit sufficient quantities of the thermostable blocking antibody (see below) to neutralize the small amounts of antigen absorbed from the respiratory membrane.

(3) Guinea pigs are readily protected from anaphylaxis by injecting an excess of preformed circulating antibody of high precipitin titer. This is called passive desensitization. If enough antibody is present it will prevent reactions to several multiples of the minimal fatal anaphylactic dose of antigen. Regardless of the amount of circulating antibody, however, a sufficient excess of antigen will produce anaphylactic reactions. The duration of this protection is short. Effective protection wanes after 48 hours. Effective use of passive desensitization has been reported in the treatment of Fall pollen hay fever and asthma, but it has not been studied sufficiently to evaluate its ultimate usefulness.

(4) A high percentage of guinea pigs which recover from sub-lethal anaphylactic reactions will tolerate several minimal anaphylactic doses for a few days. The hypersensitive state returns in two weeks or less. Isolated instances of this have been observed clinically. It is obviously not suited to planned clinical application.

(5) Specific local active desensitization is readily accomplished in guinea pigs and in man by the topical application of aerosols of antigen

to the respiratory mucosae. The co-seasonal treatment of pollen hay fever is quite effective by this method. Guinea pigs which have been thoroughly desensitized locally are not desensitized systemically. They will react fatally to minimal anaphylactic doses of antigen.

(6) Hypersensitive guinea pigs can be made refractory, unpredictably, by non-specific measures such as the injection of non-specific antisera, peptone, histamine releasing substances, anaphylatoxin, etc. Non-specific antiserum injections may prevent anaphylaxis in about 30% of a group of sensitized animals, whereas potent specific antiserum may provide approximately 100% protection.

Anaphylactic desensitization by these methods is effective and relatively simple in the guinea pig. In man, however, it is complicated by the facts that the offending antigens must first be identified, which is not always simple, and that injections of the antigens must be continued for many months or years. The ideal solution to the problem of clinical desensitization would be to find a substance which would interrupt allergic reactions in their incipiency without having to identify the allergens. In fact, Ungar has described four areas in a diagram of his concept of the pathogenesis of allergic reactions in which this might be done. Becker has postulated that it would be easy to develop specific anti-enzymes if the key enzymes responsible for the initiation of allergic reactions could be identified. With these beginnings, clarification of the mechanisms of desensitization and consequent simplification and improvement of specific therapy of allergic disorders can be anticipated with confidence.

Antibodies: The antibodies which mediate allergic phenomena have received much more searching attention than have the antigens. But important gaps remain between the knowledge of the antibodies which mediate anaphylaxis and those which mediate atopic reactions.

The antibody which mediates anaphylaxis in animals has, until recently, been considered inseparable from the precipitin which is produced readily in response to injections of antigen. Small amounts of these antisera will sensitize guinea pigs. Larger amounts will protect them from anaphylactic reactions to the immunizing antigen, as shown above. This freely circulating antibody is currently believed to be formed in plasma cells, in contrast to those which mediate the delayed types of allergic reactions. The latter are supposed to be formed in lymphocytes and monocytes. They are not found in the serum, clinically.

The problem is not as simple as that, however. Actually, recent studies have tended to confuse rather than to clarify the status of the sensitizing antibody. For example, Kabat and Sherman claim to have shown that guinea pigs and human skin can be sensitized by antisera from which all or most of the precipitating antibody has been removed by serial additions of antigen. This would imply that the sensitizing antibody has been separated from precipitin. On the other hand it has been shown in this laboratory that guinea pigs are readily sensitized by precipitated antibody which has been eluted from specific precipitates. Conversely, Sternberger claims that the antibody in soluble antigen-antibody complexes will precipitate freely with antigen after the antibody has been freed of antigen by treatment with alkali in the cold.

The nature of the antibody which desensitizes passively has received scant attention. Here again the evidence is confusing. For example, the desensitizing antibody has been found only in precipitating antisera. But several strongly precipitating antisera have failed to confer specific passive desensitization in some recent experiments. The capacity for passive desensitization is readily removed from potent antisera by the addition of antigen. Study of this antibody should be a fruitful field for research.

The antibodies which mediate the conventional clinical allergic, so-called atopic reactions have been studied extensively. The antibodies which sensitize are called reagins. The antibodies which are thought to prevent atopic reactions are called blocking antibodies.

TABLE VI

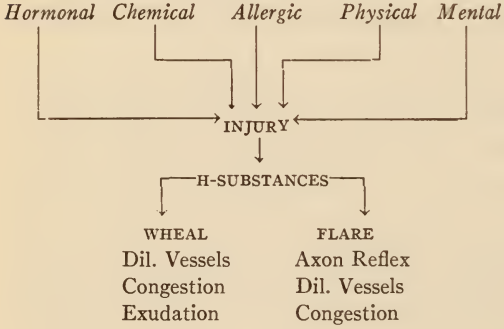
SIMILARITIES BETWEEN ANAPHYLAXIS AND ATOPIC ALLERGY

(1) Reactions are highly specific
(2) Reactions are mediated by antigen-antibody-tissue cells
(3) Antigens are apt to be inherently harmless
(4) Antibodies are readily demonstrable
(5) Active sensitization is lasting
(6) Passive sensitization is possible
(7) Passive sensitization is transient
(8) Multiple sensitizations are possible
(9) Shock tissues differ between species. Man has multiple shock organs
(10) Common pathology: exudation plus smooth muscle spasm secondary to capillary damage by the "H-Substances"
(11) "Desensitization" is possible

TABLE VII

WHEALING MECHANISM

	Acid	Bacteria		
	Alkali	Contacts		
	Atropine	Drugs		
	Bites	Foods		
	Cholines	Fungi	Cold	Life's
Estrogen	Histamine	Inhalants	Heat	situa-
Pituitary	Opiates	Parasites	Friction	tions
Thyroid	Pilocarp.	Serum	Light	
Hormonal	*Chemical*	*Allergic*	*Physical*	*Mental*



Function-wise they can be compared to the antibodies which mediate passive desensitization in guinea pigs. The chief characteristics of reagins and blocking antibodies are summarized in Table V.

The atopic syndromes, hay fever, asthma, urticaria, angioneurotic edema, and true atopic dermatitis are the closest clinical analogues to anaphylaxis. There are well known similarities and differences between them, however. The similarities are perhaps more impressive and useful in the understanding of the mechanisms of clinical phenomena than are the differences. The similarities are summarized in Table VI. The chief differences are: The seemingly spontaneous sensitization in man, the strong familial tendency, the relative inefficiency of desensitization, the prominence of reactions to ingested antigens and drugs and to environmental inhalants, and the differences in antibody behavior in man and experimental animals. It seems likely that these differences will become reconciled in the not too distant future.

Urticaria and angioneurotic edema are frequently caused by the ingestion of foods and drugs and the injection of antigens. Simple allergic reactions like those are not the only causes of urticaria, however. The various stimuli which can trigger the whealing mechanism are summarized in Table VII. Actually an allergic etiology cannot be demonstrated in a high percentage of chronic cases of urticaria and angioneurotic edema. Table VIII summarizes the incidence of the various causes of urticaria and angioneurotic edema as reported by Winkenwerder in some 200 carefully studied cases. Considerable support to the concept that urticaria and angioneurotic edema may be mediated by other mechanisms than the local anaphylactic reaction is provided by

TABLE VIII

URTICARIA AND ANGIONEUROTIC EDEMA
ETIOLOGIC CLASSIFICATION*

1. Atopic = 35%			
Foods	Pollen	Insects	Insulin
Drugs	Dusts	Serum	Liver
2. Infection = 27%			
Bacteria	Fungi	Viruses	Parasites
3. Physical = 6.5%			
Heat	Cold	Sun	Exercise
4. Endocrine = 5%			
Menopause	Menstruation	Hyperthyroid	
5. Psychosomatic = 15%			
6. Idiopathic = 12%			
7. Contact = Occasional			

* Modified from Winkenwerder

the persistence of some of the lesions, the obvious roles of friction and of temperature changes in others and the commonly observed failure of the antihistamines and the sympathomimetics to provide relief.

Hay fever: There seems to be little doubt that the classical picture of hay fever from pollens, dust, mold spores, epidermals, foods, etc. is mediated by the atopic, anaphylactic-like type of allergy. Hundreds of antigens have been identified and effective extracts for diagnosis and treatment are readily available. Many such cases of hay fever have readily demonstrable reagins which behave in accordance with the criteria listed in Table V. It is assumed that reagins mediate the hay fever in many other cases which give positive skin reactions to and are relieved by avoidance or injections of antigens to which they are sensitive clinically. This assumption takes into account the fact that methods of demonstrating reagins are crude, too crude to identify reagins even when present. This is well illustrated by recalling that high dilutions of antisera will agglutinate but passive transfer of local sensitization with dilutions of serum as high as 1:100 is quite unusual.

There are numerous clinically typical cases of hay fever in whom no evidence of a reaginic mechanism can be detected by current diagnostic means such as the history, skin tests, and antigen-elimination procedures. Some of these and many atypical hay fever-like syndromes are relieved by the effective treatment of mild sinus infections, deflected nasal septa, or the avoidance of nonspecific irritants such as tobacco and kitchen smoke, the odors of paint, moth crystals, etc.

Injections of antigens are highly effective in the treatment of hay fever and asthma from inhalants. The relief which follows a series of allergen injections is generally assumed to be due to the development of so-called blocking antibodies, the characteristics of which were summarized in Table V. The exact role of the blocking antibody in hay fever is not clear. Doubt has been voiced that the blocking antibody mediates the improvement which follows injections of pollen extracts, since the degree of relief does not parallel the titer of the antibody. For this objection to be valid it must be shown that treatment with pollen extracts provides clinical relief in the absence of blocking antibody. The most potent argument for the favorable role of blocking antibody is its capacity to prevent the customary wheal and flare reactions to intradermal injections of mixtures of antigen and reagin. This is illustrated in the sub-diagram in Table V.

There is no satisfactory explanation for the prompt relief which follows a few injections of pollen extracts during the pollen seasons, so-called co-seasonal treatment. It is comparable, superficially, to active desensitization of guinea pigs with a few sub-reacting antigen injections. But in man no detectable reduction of sensitizing antibody occurs. Most of these comments are equally applicable to asthma.

Asthma: The necessity for evaluating non-atopic etiological factors is, perhaps, more impressive in asthma than in other manifestations of clinical allergy. The conventional allergic or atopic mechanism is undoubtedly the most important single mediator of asthma. Infection, in which no feature of the reaginic type of reaction can be demonstrated, is a much less common primary cause of asthma. The mechanism by which infection causes asthma is not known, nor has it been soundly postulated.

At least seven other types of causes of asthma have been recognized. These other types of causes frequently determine the incidence and severity of attacks of asthma from reaginic allergy or infection. Occasionally, however, they seem to be primary causes. The types of causes of wheezing are listed in Table IX.

TABLE IX

CLASSIFICATION OF CAUSES OF WHEEZING

Group I	Atopy	Infections
Group II	Reflex	Psychogenic
	Physical	Chronic Lung Disease
	Non-Specific Irritants	
Group III	Cardiac	Bronchial Obstruction
Group IV	Idiopathic	

TABLE X

INCIDENCE OF CAUSES OF WHEEZING IN 100 CONSECUTIVE ASTHMAGRAMS

Allergy	90	Non-Specific Irritants	100
Infection	73	Physical Allergy	71
Cardiac	3	Bronchial Obstruction	3
Reflex	9	Psychogenic	26
Chronic Lung Disease	31		

TABLE XI

COMMON MANIFESTATIONS OF DRUG ALLERGY

1. Systemic = Chills, Fever, Prostration
2. Skin Eruptions = Many Types
   - Urticaria
   - Angioneurotic Edema
   - Erythema Multiforma
   - Erythema Nodosum
   - Erythema Scarlet
   - Fixed Drug Eruption
   - Arthus Reactions
   - Purpura
   - Herpes
   - Acneiform
   - Pruritis
   - Desquamation
   - Contact Dermatitis
3. Hematologic
   - Hemolytic Anemia—Jaundice
   - Leucopenia—Agranulocytosis
   - Thrombocytopenia—Purpura
4. Syndromes of Allergy
   - Asthma
   - Hay Fever
   - Serum-like Reactions
   - Colitis
   - Anaphylaxis

TABLE XII

MECHANISM OF ALLERGIC REACTIONS TO DRUGS

1. Drug + tissue or serum protein → Conjugated Antigen
2. Antigen in vivo → Sensitization
3. Subsequent Contacts with Hapten → Allergic Reactions
4. Sensitization Specific for:
   - Drug Alone
   - Drug + Protein
   - Related Drugs

   Specificity Determined by Chemistry of Drug, e.g.
   - *Acid Drug + Protein*→High Degree of Specificity for Acid Drug
   - *Neutral or Basic Drug + Protein*→Broader Specificity for Related Drugs

It is important to remember that asthma is not a disease. It is a syndrome to which each type of cause adds its clinical stamp to the cardinal features of shortness of breath, wheezing, cough and sputum. The clinical differences between typical allergic (atopic) and non-allergic types of asthma have been discussed elsewhere. The clinical impact of these various etiological factors on 100 consecutive cases of chronic asthma is summarized in Table X.

Drug allergy: Allergic reactions to drugs must be differentiated from toxic, pharmacological or hyperergic responses.

Table XI lists some of the more common manifestations of drug reactions. The chills, fever and prostration which are typical manifestations of the systemic tuberculin type of allergic reaction are frequently attributed to infection. The urticaria, angioneurotic edema, contact dermatitis, fixed drug eruptions, hay fever, and anaphylaxis are acceptable as allergic manifestations. The hematologic manifestations, although immune mechanisms can be demonstrated, are seldom referred to as allergic.

Table XII contains a greatly simplified summary of the mechanisms by which non-protein drugs cause allergic reactions.

Protein drugs elicit the same sorts of reactions that other proteins do, usually serum-like, urticarial, asthma or anaphylaxis. Reactions to non-protein drugs are not as predictable. Mayer feels that the type of reaction is determined by the chemistry of the drug and the route of administration. For example, drugs which are given intravenously, intramuscularly, or intraperitoneally, are quickly absorbed into the circulation and may conjugate readily with soluble serum proteins. These conjugates produce the atopic or anaphylactic type of sensitization. On the other hand, drugs which are relatively insoluble or are injected with adjuvants to delay absorption, tend to combine with the less soluble intercellular substances and to produce delayed types of allergy. This will be elaborated upon in the section on Contact Dermatitis.

Atopic dermatitis: The role of reaginic (atopic) allergy in the etiology of atopic dermatitis is extremely controversial. Those who object to the allergic etiology emphasize

the high incidence of failure of conventional allergy management; the abnormal responses of the skin to histamine, acetylcholine and dermographic stimuli (white dermographism); the thick, dry skin in uninvolved areas; the infraorbital pigmentation; and the abnormal palmar creases. Certainly "specific" allergy management of these patients is less rewarding than in the other clinical manifestations of allergy.

Serum and serum-like reactions to heterologous sera are seen much less often now than when they were more widely used. Prophylactic antitetanic serum is still used, often unnecessarily. Reactions to it are still common. Penicillin has taken the place of antisera as a common cause of serum-like reactions.

Table XIII summarizes the types of serum reactions which used to be seen so commonly.

TABLE XIII

SERUM ALLERGY

TYPES OF REACTIONS TO SERUM

Delayed = Serum Sickness
No Prior Serum Allergy
Immediate = Conventional Anaphylaxis
Sensitization from:
Natural Allergy to Horse or Rabbit
Prior Injections
Immediate = Reversed Passive Anaphylaxis
Not True Serum Allergy
Accelerated = Rapid Onset of Serum Sickness
Prior Injections → Anamnestic Response
Rapid Antibody Production
? Mild Natural Allergy
Arthus Phenomenon = Severe Local
During Serial Injections
Circulatory and Foreign Protein

Reversed passive anaphylaxis was the mechanism for many of the reactions when pneumococcal pneumonia was treated with antisera in the pre-antibiotic days. In reversed passive anaphylactic serum reactions, the antigen is provided by the disease, pneumococcal pneumonia. The antiserum injection provided the antibody to complete the allergic triad of antigen-antibody-tissues, with consequent anaphylactic shock. In this situation the patient is not sensitive to the rabbit or horse serum and could tolerate it perfectly if the bacterial antigen were not present. Along this vein, penicillin, given after the bacteria have elicited antibody responses, *may* release antigen from the susceptible organisms in sufficient quantity to combine with the antibody to produce allergic reactions. This, obviously, is not a reaction to penicillin, but to the antigen released by the penicillin. Perhaps this explains the commonly observed tolerance for subsequent injections of penicillin.

The prolonged serum sickness-like reactions to penicillin are presumed to be due to depots of antigen which continue to stimulate sensitizing antibody formation; the penicillin-antibody-tissue reactions continuing until the depot has been absorbed and disposed of. Theoretically, the incidence of penicillin sensitization can be expected to rise with increased use of long-acting penicillins. The recent introduction of penicillinase will, perhaps, lessen the importance of these reactions.

The foreign protein types of reactions to serum and to penicillin are presumably not allergic. The other types of serum reactions in Table XIII are too well known to justify further comment here.

Contact dermatitis: Contact dermatitis is mediated by an entirely different mechanism from the prompt reaginic (atopic) or anaphylactic type of reaction. Contact dermatitis is one of the two major forms of delayed hypersensitivity. The tuberculin type of allergy is the other. Studies of the mechanisms of contact dermatitis seem to have made good progress in the past decade, more so perhaps than have the investigations of the reaginic type of allergy. The key features of the sequence of events in the development of the contact type of sensitization are shown in Pillsbury's diagram in Fig. 1.

Mechanism of sensitization: Non-specific and specific factors must operate in order to overcome the normal resistance to sensitization. The allergens are usually haptens which combine with the skin proteins in the basal cells or the intercellular substance. The union between skin protein and hapten may be physical, not

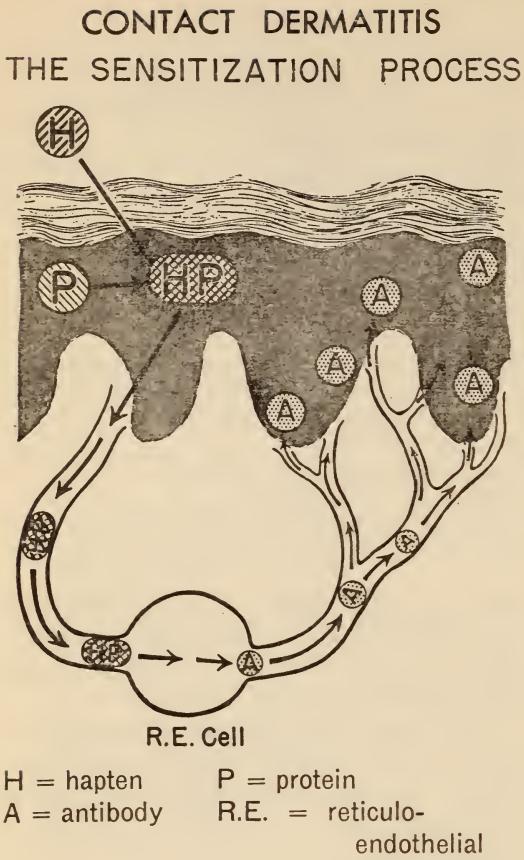


FIG. 1. The mechanism of contact sensitization. (From Pillsbury's Dermatology).

chemical. The combination must be "trapped" in the epidermis. *Non-specific factors* which promote adequate contact between the hapten and the epidermis are pressure, friction, sweat, nonspecific irritation, a shift of the pH towards alkalinity. The function of the nonspecific factors is to remove the protecting oily layer and the superficial keratin layer. Oil soluble haptens seem to penetrate readily. Heavy metals and dyes are fixed readily to the skin proteins when the protecting substances have been penetrated.

Specific factors. The point of contact with the hapten is the epidermis. Experimentally, the hapten contact can be made with the epidermis directly or by injecting the hapten intraperitoneally with adjuvants. Clinically, the usual sensitizing contact is with the epidermis directly. The duration of the contact seems to be important. It probably varies with different haptens. With dinitrochlorbenzine the sensitizing contact with the skin must be at least one day. The concentration should be less than that which irritates the skin. With that drug 0.1–1.0% have been effective. It takes about 6 to 9 days for sensitization to occur and 12 to 15 hours for the reaction to appear after the hapten comes in contact with the sensitized skin.

The evidence seems quite clear that sensitization varies in degree with the allergen, the duration and number of exposures. Once established sensitization may last for weeks or years.

Antibodies are presumed to be formed in regional lymph nodes and in the central portions of the reticulo endothelial system, whence they are carried back to and are fixed in the deeper layers of the epidermis. Although the antibodies, as such, have not been identified, the inference that they mediate contact dermatitis is strong. Some of the evidence follows: Contact dermatitis can be passively transferred by monocytes, lymphocytes, transfusions of whole blood and by the cells of peritoneal exudates from skin-sensitive animals. Normal skin, grafted to a skin-sensitive subject, will develop sensitization comparable to the surrounding sensitive skin. A parabiont, if adequately prepared, will soon share the skin sensitization with the sensitized partner. On occasion, blister fluid will transfer the contact dermatitis type of sensitization, presumably mediated by the cells in the fluid. Monocytes are believed to be increased in the circulating blood, while sensitization is developing.

In spite of the inability to demonstrate the mediating antibody, so far, there is considerable evidence that points to the allergic nature of contact dermatitis. This evidence is summarized briefly in Table XIV.

Re-exposure to the allergen may maintain or renew sensitization or it may desensitize. The factors involved in desensitization have not been studied adequately. Local application of the allergen to highly sensitized skin may result in generalized dermatitis. This tends to

TABLE XIV

ALLERGIC NATURE OF CONTACT DERMATITIS

(Sulzberger)

Highly Specific
Patch Tests Reproduce Disease
Patch Tests may Sensitize
Incubation Period Follows Sensitizing Contacts
Flare-ups Produced at Will with Repeated Contacts
Temporary Desensitization May Follow Reactions
Reaction Time after Contact = 24–48 Hours
Reactions = { Non-Pharmacologic = Allergic / Non-Toxic }
Minute Contacts May Cause Alarming Reactions
Sensitization is Lasting
Symptoms Subside if Contact Stops
Multiple Sensitizations are Common
Shock Organ is Epidermis

be followed by a short period of desensitization. The spontaneous desensitization which is observed in industry following continued exposure to the allergen is called hardening. It should be possible to determine schedules by which graded exposures to individual allergens would lead to tolerance. It is surprising that more information is not available on this subject.

SUMMARY

A simple practical concept of the immunological and other mechanisms which mediate the common allergic disorders has been presented. The roles of antigens, antibodies and tissues in anaphylaxis, desensitization, serum reactions, asthma, hay fever, urticaria, angioneurotic edema, atopic and contact dermatitis have been emphasized. The roles of non-immunological factors in the pathogenesis of these disorders have been considered. The aim has been to provide a working rather than a detailed knowledge of the subject.

Attention has been called to the deplorable lack of instruction in our medical schools and hospitals in these important common causes of illness.

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2. RATNER. Allergy, Anaphylaxis and Immunotherapy., (Williams & Wilkins)
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9. American Journal of Medicine, Vol. XX., (Series of Papers on Mechanisms of Allergy)
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Studies on the Properdin System in Transplantable Cancer[1]

By PETER A. HERBUT, M.D.[2]

PRESIDENT RHOADS, Members of the Mütter Committee, Fellows of the College of Physicians of Philadelphia, and Guests. It is indeed a great honor to have been asked to deliver the Seventy-First Annual Thomas Dent Mütter Lecture this evening and thus to be included among such legendary notables as Samuel W. Gross, Harvey Cushing, William J. Mayo, George W. Crile, Rudolph Matas, J. Chalmers Da Costa, James Ewing, Evarts Graham, Edward Archibald, H. Gideon Wells, Edward D. Churchill, and Balduin Lucké—to mention but a few of the lecturers of the past. Although the bequest of Thomas Dent Mütter in reference to the Lectureship specifically mentioned "... some point or points connected with surgical pathology", the Committee on Scientific Business of the College has been most generous in allowing essayists to select a wide range of topics not directly related to surgery. The subject of my talk falls into this category.

The easiest way to approach any subject is to start at the beginning and this I propose to do this evening. At Jefferson we have the Elizabeth Storck Kraemer Memorial Foundation, started some thirty years ago by Dr. William H. Kraemer and sponsored primarily by the now late Mr. Pierre S. duPont and his brother the late Mr. Lammot duPont. The main purposes of the Foundation are (1) to provide facilities for the diagnosis and treatment of cancer in patients, (2) to search for chemotherapeutic agents that might be useful in the treatment of neoplastic diseases, and (3) to investigate the causes of cancer. It is in connection with the latter two purposes that the work which I shall report on tonight is being conducted.

For years we have been screening chemicals for tumor inhibitory properties against a variety of animal tumors. While some of these agents showed considerable experimental promise, it became increasingly apparent that transferring such procedures to humans met with universal failure. Thus we, as others, wondered whether we might not do better by using, if possible, a human tumor grown in experimental animals. About this time, Toolan (12), using and extending Murphy's observations of 1914 (9), succeeded in growing several human tumors in irradiated and cortisone-treated rats and mice. Employing a similar technique, we (1) attempted to develop our own human-tumor-rat combination by using weanling Wistar rats and human tumors removed freshly and aseptically in the operating rooms. Of 220 tumors implanted, 206 were cancers and of these only one (HR 132) grew. This was a carcinoma of the ascending colon removed from a seventy-seven year old woman on December 29, 1954. Transplanting the growth every 10 days into weanling Wistar rats exposed 24 hours previously to 300 r total-body irradiation, the tumor is now in the 120th generation and still growing well.

Because of these results we, as others before us, were impressed with the natural resistance of animals not only to heterologously transplanted tumors but also to heterologously transplanted tissue of any kind. Seeking an answer to this phenomenon, we were unable to find any satisfactory explanation in the literature where only such vague statements

[1] Thomas Dent Mütter Lecture LXXI, College of Physicians of Philadelphia, 1 October 1958.

[2] Professor and Head of Department of Pathology and Director of Clinical Laboratories, Elizabeth Storck Kraemer Memorial Foundation, Jefferson Medical College of Philadelphia, Philadelphia 7, Pa.

as "immune reaction" (7) and "natural and acquired immunity" (13) were promulgated.

About this time, there appeared in the literature two (as we at that time thought) important and seemingly unrelated discoveries. The first was that of Pillemer (10). In 1954 he isolated a new serum protein—a euglobulin which he called properdin (Lat.—perdere = to destroy). In the presence of complement and Mg^{++} this substance kills bacteria, inactivates viruses, lyses certain types of erythrocytes, and kills some protozoa. Among other properties, it is depleted in rats subjected to total-body irradiation. This lowers the resistance of the animals and thus renders them susceptible to infection. The second discovery was that of Kidd. In 1953 he (8) demonstrated that normal guinea pig serum contains a substance (thought by him to be a protein) which causes regression of subcutaneously implanted Gardner lymphosarcoma 6C3HED and lymphoma II in mice. Thus, because (a) total-body irradiation to rats causes depletion of properdin, (b) total-body irradiation to rats is necessary to destroy "something" in order that human tumors may grow, and (c) a "protein" in normal guinea pig serum destroys lymphosarcoma in mice, we (1) *theorized* (a) that properdin and the "protein" in guinea pig serum may be one and the same and (b) that properdin may be the natural tumor inhibitor in the animal body. Having thus advanced a possible explanation, the next step was to investigate the hypothesis. This we did by using two systems—the human tumor HR132 in Wistar rats and the Gardner lymphosarcoma 6C3HED in C3H mice.

HUMAN TUMOR HR132 IN WISTAR RATS

You may or may not know that it is difficult to manufacture properdin and that it is virtually impossible to purchase the protein on the market. The properdin level in animals, however, is influenced by intravenous injection of *zymosan*—an insoluble carbohydrate complex derived from yeast cell walls. In fact, it was by the use of zymosan that properdin was isolated in the first place. By using zymosan, Pillemer and Ross (11) in 1955 showed that intravenous injections of 5 mg. of the compound into animals resulted in an immediate fall of properdin titer to 30% of normal with a subsequent rise to 300% of normal. Injections of 125 mg. of zymosan resulted in a fall to 20% of normal with a subsequent rise to 75% of normal. Using this work as a basis we (2) implanted tumor HR132 subcutaneously into weanling Wistar rats—some exposed to total-body irradiation and others treated with zymosan, and obtained the following results (160 rats in each group): nontreated—19 growths, 300 r total-body irradiation—136 growths, 1 injection of zymosan—58 growths, and 4 injections of zymosan—90 growths. Pathologically, irradiated animals showed lymphoid atrophy whereas zymosan treated animals disclosed reticulo-endothelial and lymphoid hyperplasia.

These results, we thought, gave credence to the theory that the properdin system might be involved in the natural resistance of animals to heterologously transplanted tumors. The only defect in the experiment was that properdin levels on sera from the rats used were not determined. This was so because the assay was too complicated for us to undertake and because we had not yet established a liaison with Doctor Pillemer. Later, this experiment (using a total of 296 rats) was repeated (3) not only for the purpose of checking the number of tumor "takes" but also for the purpose of determining the *properdin titers* on the sera of the animals. While the growth of the tumors paralleled rather closely that of the tumors in the first experiment, the properdin titers showed no constant relationship to tumor "takes" inasmuch as some were slightly depressed, others were normal, and others still were elevated.

Despite the inconstant correlation referred to above, it was nevertheless deemed advisable to attempt to restore the *properdin* levels and resistance of the animals treated with 300 r or zymosan by the injection of properdin itself. Since the amount of properdin

(human) we were able to obtain was limited and since more consistent results, both with regards depression of properdin levels and growth of tumor, were obtained with irradiated animals, irradiation rather than zymosan was selected to cause depression of the properdin system. In the experiment (using a total of 60 rats) which followed (3), the properdin titers of the sera of irradiated animals were raised by the injection of properdin almost threefold and yet such elevations had no effect upon inhibiting the growth of HR132.

From the foregoing it is apparent (1) that both irradiation and zymosan destroy the resistance of weanling Wistar rats to HR132, (2) that such treatment has no consistent or uniform influence on the properdin titer of serum from the rats, and (3) that restoring the blood properdin levels in irradiated rats with *human* properdin does not restore their resistance to HR132. It is thus *concluded* (1) that the natural resistance of weanling Wistar rats to the transplantable human carcinoma of the colon HR132 is not mediated through the properdin system, (2) that the properdin was administered at the wrong time, (3) that pretreatment with roentgen rays alters other critical factors rendering the administered properdin ineffective, or (4) that heterologous (in this case human) properdin is ineffective even though it can be detected in the animals by zymosan assay.

GARDNER LYMPHOSARCOMA 6C3HED IN C3H MICE

The second tumor-animal combination used in the investigation was that of the Gardner lymphosarcoma 6C3HED carried in C3H mice. Its purposes were twofold—(1) to study the role of the properdin system in the growth of this transplantable tumor and (2) to determine the nature of the tumor inhibitory principle (TIP) in guinea pig serum—whether properdin or some other substance. In the experiments, most of the sera were obtained in the lyophilized state from Cappel Laboratories, West Chester, Pa. The animals weighed about 20 g. each and were purchased from the Jackson Memorial Laboratories, Bar Harbor, Maine. This tumor-mouse combination is excellent because the tumors take and ultimately cause death in 100% of cases. They never regress spontaneously. To date, we have used well over 3000 animals (4, 5, 6).

One series of experiments consisted of studying the tumor inhibitory *effects* of *combinations* of *guinea pig serum*, *properdin*, *$MgSO_4$*, *rat serum*, *irradiation*, and *zymosan* (4). The findings disclosed (1) that tumor growth in nontreated mice was excellent, (2) that the tumor inhibitory response of adequate doses of guinea pig serum was also excellent, (3) that administration of properdin in 25 and 50 unit doses showed no tumor inhibitory response, (4) that properdin given in combination with below tumor inhibitory amounts of guinea pig serum, in an attempt to supply extra complement and perhaps Mg^{++}, disclosed no adverse effect upon the growth of the transplanted tumor, (5) that the addition of $MgSO_4$, to supply extra Mg^{++}, to the combination of properdin and guinea pig serum revealed no tumor inhibitory response, (6) that rat serum, which contains a high properdin titer, combined with below tumor inhibitory amounts of guinea pig serum, to supply extra amounts of complement and perhaps Mg^{++}, did not affect tumor growth, (7) that normal nontumor bearing mice showed as much as a threefold increase in properdin levels when treated with normal or heated guinea pig serum, and (8) that depression of properdin titers in the circulating blood by total-body irradiation or administration of zymosan did not impede the tumor inhibitory action of guinea pig serum. In the same series of experiments, determinations of properdin titers on sera from animals in the various groups showed that the properdin levels varied inversely with the growth of the tumor. In other words, the titers were low when the tumors grew and were elevated when the tumors regressed—regardless of the type of treatment. These *results* tend to *indicate* (1) that TIP in guinea pig serum is probably not properdin, (2)

that the properdin system in C3H mice is stimulated by the injection of normal guinea pig serum, and (3) that the properdin titers of mouse sera vary inversely with the growth of tumor 6C3HED. Such fluctuations, however, may represent simply a reflection of tumor growth rather than its determinant.

The next series of experiments were designed to *attempt* to *identify TIP* in guinea pig serum (5). The findings disclosed (1) that TIP was not destroyed by heating guinea pig serum at 66°C for 30 minutes, (2) that guinea pig serum depleted of complements I, III, and IV as well as depleted of properdin still retained TIP, but depleted of complement II lost TIP (not significant because latter is made from the precipitate obtained in depleting guinea pig serum of complement I), (3) that the supernatant from guinea pig serum centrifuged at 35,000 G. at 2°C for 3 hours retained TIP but the pellet (containing lipopolysaccharide) did not, (4) that serum from tumor bearing and from nontumor bearing mice treated with guinea pig serum (and presumably containing a high titer of mouse properdin) showed no tumor inhibitory properties, (5) that a *Streptococcus hemolyticus* isolated from the guinea pig serum disclosed no tumor inhibitory properties, (6) that TIP was not extractable by treating guinea pig serum with ether, acetone, or dimethylformamide but was extractable with methanol, (7) that TIP in the methanol extract is inactivated by bringing the pH below 6 but is not inactivated by raising it above 7, (8) that TIP is present in guinea pig serum and plasma and is not carried in the erythrocytes or the products of erythrocytes, and (9) that electrophoretic studies on guinea pig serum and methanol extract of guinea pig serum seem to indicate that TIP is present in the globulin fraction. These *results* tend to *show* (1) that TIP is not complement, properdin, or lipopolysaccharide, (2) that TIP is present in guinea pig serum and not in the erythrocytes or their products, and (3) that TIP is extractable with methanol. Further studies on the identification of TIP are currently under way.

OTHER ANIMAL SERA

A final series of experiments was designed to determine whether TIP is peculiar to guinea pig serum or whether serum from other animals may also contain the principle (6). This information would be of particular interest in connection with larger animals, such as bovine and sheep, which could serve as far better and less expensive sources of serum for investigative purposes than does the guinea pig. In the experiments, complete inhibition of growth of tumor 6C3HED was obtained with guinea pig serum; slight and inconstant retardation was shown with sheep and human sera, and no inhibition was detected with rat, chicken, dog, bovine, horse, calf, and rabbit sera as well as with sheep and human plasma. If these sera do contain TIP its detection must await concentration or fractionation procedures.

SUMMARY

The investigation of the possible role of the properdin system in transplantable cancer was studied in connection with the transplantable human carcinoma of the colon HR132 in weanling Wistar rats and the Gardner lymphosarcoma 6C3HED in C3H mice. The results indicate the following:

(1) Animals can be conditioned to heterologously transplanted tumors by irradiation, cortisone, or zymosan.

(2) Irradiation and zymosan, in doses necessary to do this, depress the properdin level in Wistar rats but slightly.

(3) Restoration of the properdin level with *human* properdin in irradiated rats does not restore their resistance to heterologous transplants.

(4) The operating mechanism is thus either not through the properdin system or the conditions of the experiment were not correct.

(5) The tumor inhibitory principle (TIP) in guinea pig serum has so far been shown to

be present only in guinea pig serum and not in other mammalian sera or in chicken serum.

(6) TIP is present in the fluid and not the cellular portion of the guinea pig blood. It withstands heating to 66°C for 30 minutes, is extractable with methanol, and probably exists in the globulin fraction of the serum. It has not yet been identified but has been shown not to be complement, properdin, or lipopolysaccharide.

(7) Properdin levels of sera from C3H mice vary inversely with growth of tumor 6C3HED, and

(8) This fluctuation is probably an indicator of tumor growth rather than its determinant.

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Technical Cooperation—A Challenge[1]

By EUGENE P. CAMPBELL, M.D.[2]

MR. CHAIRMAN, Mrs. Hitchens, members of the Pennsylvania Public Health Association, College of Physicians of Philadelphia and guests; it is a great pleasure for me to be here this evening. To return to Philadelphia where my wife and I spent three of the most pleasant and most productive years of our lives; to return to honor the memory of my former boss, the late Colonel Arthur Parker Hitchens; to return with this great privilege of leading a period of thinking and discussion on a topic that was of great interest to "The Colonel"; to renew and strengthen the bonds of friendship and collaboration with you and others in the Philadelphia area; all of these intense human features have been uppermost in my mind as a great personal honor and privilege.

Through Dr. Claude Brown, Dr. John Lentz, Dr. John Hanlon, Dr. Fred Rogers, Dr. Murial Robinson and others, I have learned of and had the privilege to read much of the material on "The Colonel's" life and work that has been presented in earlier years. I would like to contribute a glimpse of my personal relationship with him; tell you how his life fits into the career of one of his boys, and discuss with you "Technical Cooperation—A Challenge" as fruit from one person's career which had its roots in this intensely memorable relationship.

During my medical internship and assistant residency at the Baltimore City Hospital, under the inspiring leadership of the late Dr. Thomas Boggs, I had my sights on internal medicine as a career. Without the financial resources to continue, I turned to a course of trying to keep body and professional objectives together and had the good fortune to secure an assignment at the Walter Reed Hospital under the direction of the distinguished and lovable Colonel Arturo Carbonnel, Chief of the Communicable Disease Service.

During nearly a five year period, I struggled with the problems of serum sickness, prontosil, sulfanilamide and other emerging chemotherapeutic measures in the treatment of the infectious diseases. It was during this period that I had many inspiring contacts with Colonel Hitchens, who was Chief of the Bacteriology Laboratories at the Army Medical Center. Our clinical difficulties with the management and treatment of epidemic meningococcal meningitis were eased by the thoughtful, careful and expert considerations of "The Colonel". He opened up an entire area of interest for me, leading to such elements as Bergey's manual, George Dunham, the Society of American Bacteriologists, Dr. Sarah Branham, 'Tularemia' Francis,[3] Rolla Dyer and many others.

After these five years of experience in the communicable disease field in association with Arturo Carbonell, Bert McKie, George Dunham, and especially "The Colonel", I began to turn my career interests toward public health.

When Colonel Hitchens was appointed Professor of Public Health and Preventive Medicine at the University of Pennsylvania, he invited me to join the staff as Assistant Professor of Epidemiology. This period, from 1939 to April 1942, is very vivid in my memory and full of the type of intimate and personal relations of a junior member of a staff led by a dynamic, scientifically fearless, warm and understanding person. The kaleidoscopic recollection of persons and events during these

[1] Arthur Parker Hitchens Lecture III, Joint Meeting of the Pennsylvania Public Health Association and the College of Physicians of Philadelphia, Section on Public Health, 15 October 1958.

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three years brings to mind the staff of the School of Public Health, the Bureau of Laboratories of the State, the Medical School, Philadelphia General Hospital, the City Health Department, the State Health Department, and finally the entry of the U S. into World War II.

In addition to the Colonel's influence on my decision to join the staff in Philadelphia, he had shared with me some of his great interest and experience in the health problems of other countries, principally the Philippines. I had learned in detail some of his experiences with Colonel Siler, during their studies on dengue. He spoke often of his Philippine work as a participant in the development of the School of Sanitation and Public Health which today is the Institute of Hygiene of the University of the Philippines. When General George Dunham and Colonel Albert Driesbach invited me to join with them in the establishment of cooperative health programs in the Republics of Central and South America, little did I realize that I would be taking up an activity which would engage me for so many years. Much of my present interest in and enthusiasm for international health, however, stems directly from the years I was fortunate to have with Colonel Hitchens.

The presence of Mrs. Hitchens here this evening gives me an additional opportunity to say that I am proud to be able to share with you and with her this short account of one very small facet of "The Colonel's" full life.

I wish to devote the balance of the time available to me to describe for you what our Government is doing in the field of Technical Cooperation in health and to analyze with you some of the features of the inter-personal relations between professional and technical personnel from different cultures that make possible a degree of success in this field.

Technical Cooperation is best known as Point 4. It accounts, on the one hand, for about 10 percent of ICA's funds and, on the other hand, about 85 percent of ICA's employees. These professional and technical personnel are engaged in cooperative activities with the professional and technical personnel of 60 nations around the globe. It represents a series of far flung operations vital to the interests of the people of the world for whom the problems of food, shelter, health, education and welfare dominate all other considerations.

Some of you may have read in the newspaper how ICA quickly responded to the epidemic of cholera and smallpox in Pakistan. We made large amounts of needed vaccines available and joined with our Public Health Service to send a team of epidemiologists to assist our staff in the cooperative program in Pakistan and their national colleagues control these two diseases.

More recently a sharp epidemic of what was feared to be encephalitis appeared in Thailand and upon request we were able within a few days to send Dr. Hammond of Pittsburgh to Bangkok. In a few weeks he identified this epidemic as hemorrhagic fever similar in some respects to dengue rather than the more serious Japanese B encephalitis.

At a recent meeting in Dallas, Texas, Mr. Smith, our Director, made the following points: Since World War II, twenty new independent nations have come into being. Their total population is about 750 million, more than three times that of the U. S. and about one-fourth of the world's total. Each of these nations has emerged from colonial or dependent status to independence.

Coupled with strong feelings of nationalism, these people have an intense desire to achieve better living standards, more food, improved health and to share in the better life. To these people poverty, disease, ignorance and the daily struggle to secure food and water have been their inescapable lot. Now they know there is another way to live. Their desire to improve themselves is the most powerful force in the world today. The pulse of progress is quickening for millions and it has been called "the revolution of rising expectations."

We Americans are big hearted. We hear about the above-mentioned conditions and hope they are not true. When the facts are

confirmed we follow a traditional American custom of wanting to help the less fortunate. We are fundamentally generous. Helping one's neighbor or joining a group to help an unfortunate colleague is a way of life among Americans.

We are faced with a dilemma, however, when the object of our generous feeling is 10,000 miles away and, as individuals, find it nearly impossible to help except to send food, clothing and such items as one identifies with disasters.

Herein lies one of the most important reasons for having the person to person approach of Technical Cooperation. The millions seeking independence do not like to accept gifts—or charity—from others, no matter what the motives may be. They prefer to stand on their own feet through their own efforts. They have inherent in themselves the human potentials to achieve economic development and thereby a greater measure of independence but they lack experience and the knowledge of how to get this process in motion. The ICA's approach to this matter is implementation of Technical Cooperation in many fields, including health.

Some of you will ask about the role of the World Health Organization, the Pan American Sanitary Organization, United Nations Children's Fund and other United Nations connected organizations. Historically speaking, we have grown up with the United Nations organizations. Aside from coordination problems, the world need is so vast that our respective roles have been principally complementary.

It should be pointed out that the basic difference between our approach and that of the World Health Organization can be found in the meaning of the terms multilateral and bilateral. The activities carried out by the World Health Organization are multilateral in that they are authorized by a congregation of the 88 member nations, each nation having its vote.

The activities carried out under the bilateral approach are authorized by a joint agreement between the United States and another country. We have a coordinating mechanism with the multilateral organizations which operates on a daily round of business basis. Problems always exist but none of them is insoluble.

TABLE 1

Approximate United States Financial Contributions to International Health Activities

(*Fiscal Year 1958*)

Source of Funds	International Health Activities		
	Bilateral	Multilateral	Total
Mutual Security Act:			
ICA	$42,492,000	$7,000,000	$49,492,000
UN Children's Fund		7,801,500	7,801,500
UN Expanded Technical Assistance		2,457,900	2,457,900
Total	$42,492,000	$17,259,400	$59,751,400
Public Law 643			
World Health Organization		$4,666,480	$4,666,480
Pan American Sanitary Organization		1,914,000	1,914,000
Public Law 410			
Public Health Service		$137,000	$137,000
National Institutes of Health	$1,987,325*	300,000	2,287,325
Grand Total	$44,479,325	$24,276,880	$68,756,205

* Estimated.

THE DIMENSIONS OF THE ICA BILATERAL HEALTH ACTIVITIES

I would like to give you some idea of the dimensions of the international health activities of our Government (Table 1). The United States contributions to the World Health Organization (WHO) and Pan American Sanitary Organization (PASO) represent our share of, or in a sense our dues to, the total cost of these growing and important organizations. A portion of the U. S. contribution to the United Nations Expanded Technical Assistance program is for health purposes and therefore administered by the WHO. The U. S. contribution to the UNICEF is used for materials and supplies to be made available to advance the welfare of mothers and children around the world.

The largest U. S. contribution to international health is made by the ICA through the bilateral cooperative health programs with activities in forty-four countries (see Map 1.).

Each of these countries has requested help from the United States in some aspect of public health and each agreement to cooperate is incorporated into a formal bilateral understanding. In forty of these countries we have 343 professional and technical personnel stationed in groups of from four to 15. Each person or group is engaged in collaboration along the lines requested and a summary of these activities follows in Figure 1.

Throughout the less developed areas of the world the principle causes of death and illness are still the infectious agents, in contrast to the degenerative processes characteristic of the more developed areas. Since nearly all our requests come from the lesser developed or newly developing countries, we find that most of ICA's resources, in personnel and funds, are employed in the control of specific infectious or communicable diseases. Inevitably much of our collaborative work is in the field of environmental sanitation and in the development of rural community health services.

One aspect of our work involves the provision of fellowships for advanced studies to properly qualified health personnel. The largest number are granted for training within the recipient's home country. Such periods of study are often carried out in Universities. More often they involve special courses designed to meet the specific needs of nurses' aides, sanitary inspectors, engineers, nurses and doctors in relation to the cooperative health work. A substantial number of fellowships are granted for study in a neighboring country and finally some are brought to the United States for advanced study. Table 2 shows the total number and classification of the fellowship grants made to students (participants) coming to the United States during a four-year period, 1954 through 1957.

One aspect of the bilateral health programs which should not be overlooked is represented by the financial contributions which collaborating countries are willing to make available to the cooperative health work. (Figure 2.) During a 15-year period the Latin American countries have not only contributed a substantial and ever increasing amount of funds to these cooperative health activities, but their portion of the total cost is increasing. Although the programs in South Asia and the Far East are younger in age, certain Ministries of public health with which we have program agreements, notably Iran, India and the Philippines, show ever increasing appropriations for their work.

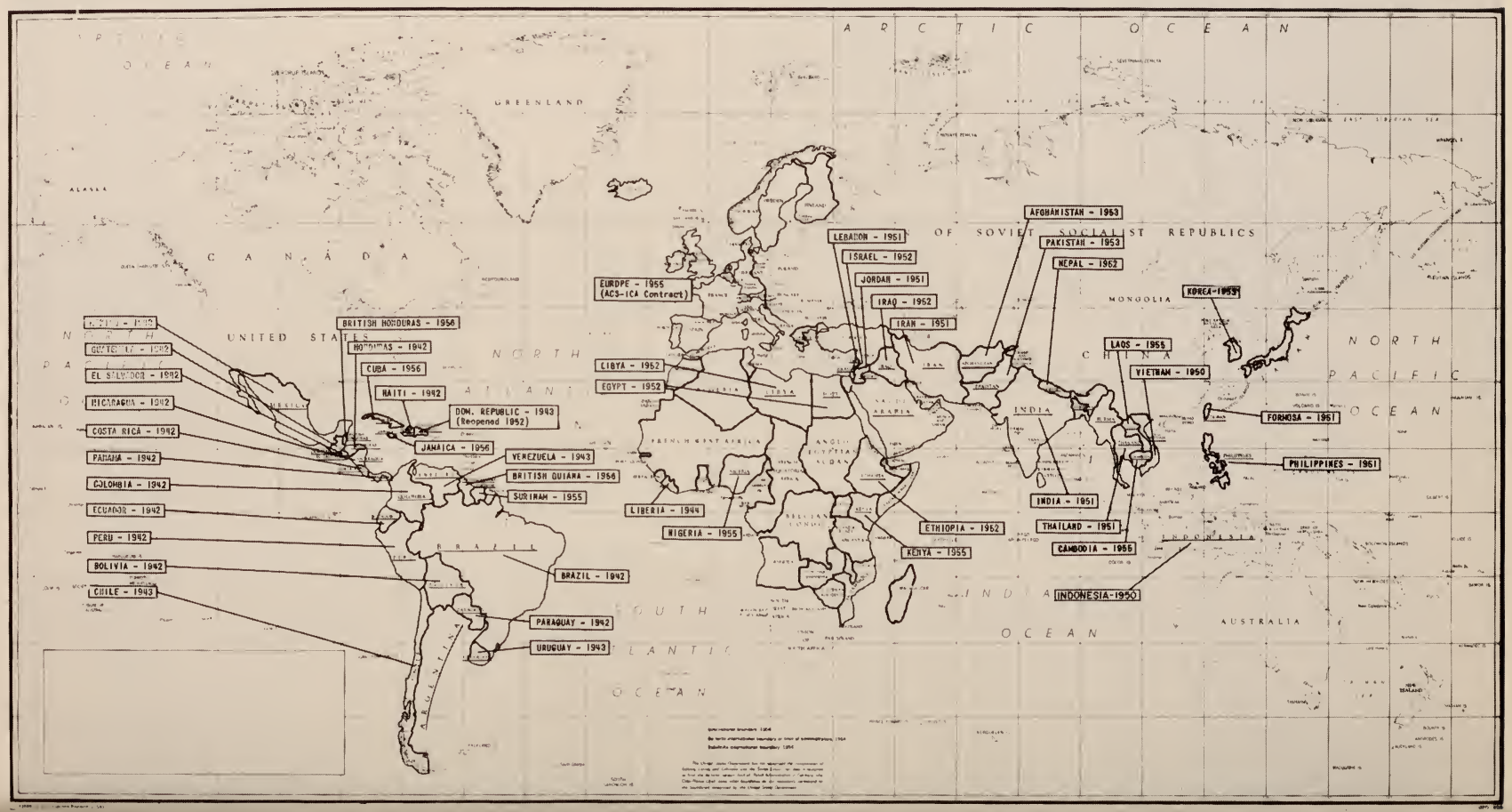
INTER-PERSONAL RELATIONS IN BILATERAL PROGRAMS

Basically three types of working relations exist between the U.S. professional and technical personnel and those of the host country as follows:

1. A host country requests the services of a specialist to help solve some pressing health problem. The U. S. pays the salary, travel and part of the perquisites. The host country pays some of the perquisites and provides working arrangements and local travel. The

Map 1

ICA HEALTH PROGRAMS—WHEN STARTED



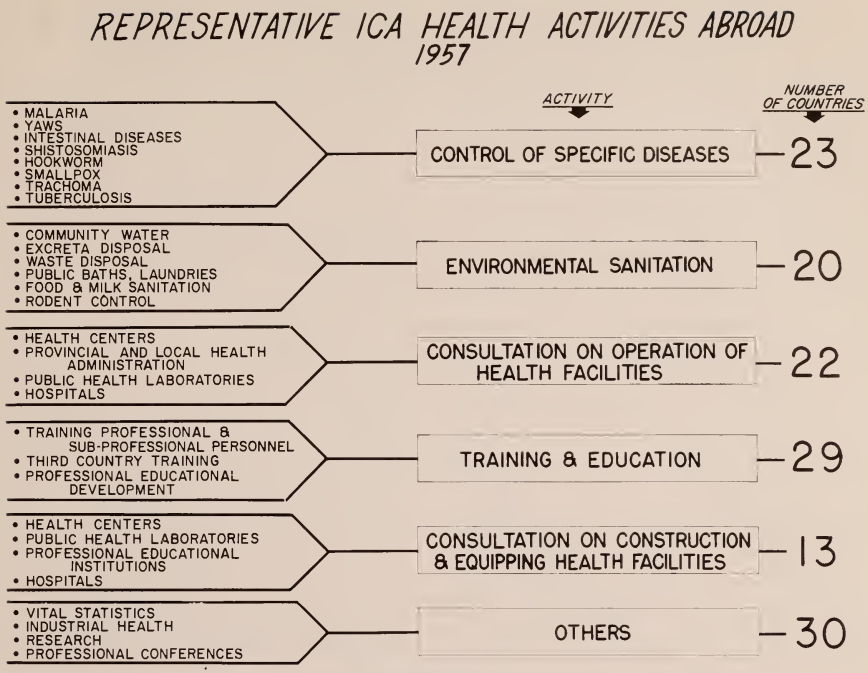
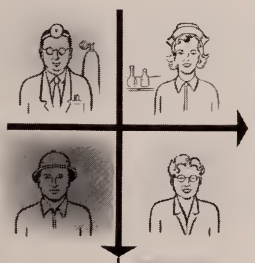


Fig. 1

TABLE 2



PUBLIC HEALTH PARTICIPANTS

	Physicians	Nurses	Sanitary Engineers	Other	Total
1954	98	40	38	47	223
1955	91	57	52	53	253
1956	160	60	57	93	370
1957	137	68	61	77	343

American works with the nationals of the country requesting his services. He investigates a problem and leaves a report. His relationship is essentially that of an investigator.

2. A group of American health personnel are attached to the United States Operation Mission[4] in a country and are assigned to a variety of projects which have been agreed

[4] The International Cooperation Administration (ICA) has field Missions located in about 60 nations of the Free World. These Missions are called United States Operations Missions (USOM).

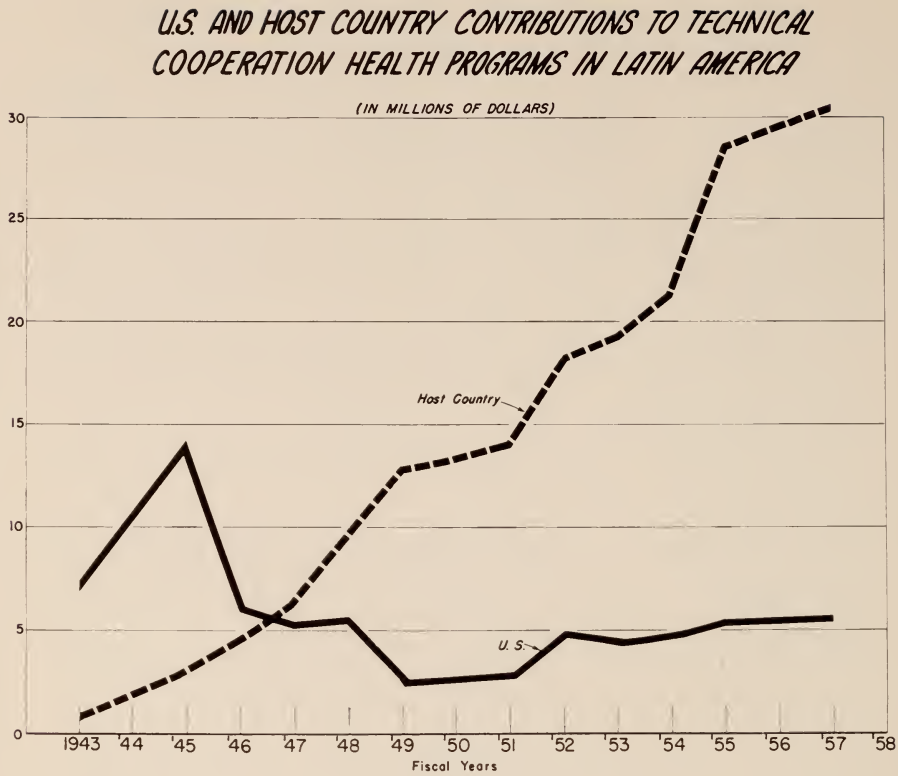


Fig. 2

to between representatives of the U. S. and the host country. The personnel work as technical advisors to the USOM and to the host country counterparts without administrative participation or control of the funds involved. They sometimes engage in limited operational activities along with their national counterparts. Contracts between ICA and Universities[5] or State Health Departments have been used as a device to provide personnel and program direction in a few instances.

3. A group of American health personnel are joined to a group of host country health personnel by a formal agreement which states the objectives, the program, fields of activities, the amounts of financial resources, conditions for employment of personnel, etc. Such a group is usually identified as an integral part of, and subordinate to, the host country Ministry of Health. This unit administers and controls the joint funds used to carry out only those projects agreed to under the formal health agreement. Contracts as just mentioned plus joint fund contracts with Universities or other sources of personnel are employed. The American in these circumstances assumes the role of a professional colleague.

All three of these administrative patterns exist within the framework of bilateral cooperative programs. Each type has its limitations and advantages. Each type is now employed by ICA under varying circumstances. Each has been utilized to meet certain objectives within the overall framework of technical cooperation. During the years some programs have changed from one type to another.

Whichever pattern is employed, success, in the last analysis, depends upon a relationship between people. The role of the American

[5] ICA has contracts with 56 American Universities which in turn have personnel in 35 foreign countries. Six of these contracts are in the field of health.

technician, therefore, is of the utmost importance. The interaction or interpersonal understandings, in the final analysis, determine the degree of success achieved in any cooperative venture.

DURATION OF ASSIGNMENT

The proper length of service in a cooperative program is of great importance to the success of the activities engaged in. Americans are usually assigned on a two or four-year tour to work with selected local health personnel to promote and help develop important health activities. Under certain circumstances (1 and 2 above) he acts in a purely advisory or counterpart capacity; in others (3 above) he shares in the administration of projects and likewise shares in the results.

A few American personnel are what we call "short-termers". They are usually specialists and are brought into action because of a special need that neither the host country nor the regularly assigned Americans can supply.

We Americans often think that a two-year assignment abroad is long enough. In our ICA experience, however, we find that in two years a person is just becoming oriented to the local cultural scene. We consider four years an average length of tour and many of the most successful tours are six, eight or more years. Programs with only short-term American personnel seldom afford enough time to get "in touch" with the local problems. Success is limited and costs are relatively high. Considering our objectives in building strong local institutions in health, continuity becomes an important factor. Dr. Paul Russell aptly remarked that "perfection and speed in any line of human endeavour are expensive. There must be constant striving for continuity of modest effort."

THE CONSULTANT'S ROLE AND THE SIGNIFICANCE OF CONSULTATION

This means many things to many people and in order to avoid the impression that I am indulging in a by-play in semantics, I wish to state that these terms are used in their broadest sense. In international cooperation we feel it is a two-way process so that everyone is both consultant and consultee in the normal day-to-day activities. For want of a better or more specific term, we tend to use consultation to categorize this inter-personal relationship between professional personnel.

We assume, of course, that the American candidate for this role comes to us with high professional and technical competence including acceptable personal and social qualities; that he has a sufficiently wide experience in public health and that he either has the capacity or can learn to communicate his ideas and knowledge in an acceptable manner.

If our consultant is engaged primarily in demonstrations, or if he works on special studies, or becomes part of a group or devotes most of his time to the day-to-day work of public health, he will have a wide range of experiences which will indeed challenge him to do his best.

An American nurse and her Iranian counterpart were asked to pay a visit to a small village on the shore of the Caspian Sea named Ojahsar. On the day they arrived, the village midwife indicated that she had a delivery to perform and asked the nurses to accompany her.

I visited this village on October 1, 1952—40 days after the nurses' first contact. The Mayor told me with great delight that the child delivered with the help of the Point 4 nurses was still alive and was the first child born that year that had lived beyond 10 days.

Furthermore, he indicated that for the first time in many years his villagers had sufficient rice to eat because the cooperative health team had sprayed all their houses with DDT. Thus, instead of nearly everyone being sick with malaria during harvest time, everyone was well and able to gather in the rice.

Under such conditions the American carries a certain amount of responsibility to perform with subordinates, equals and superiors in his area of professional competence. This relationship often brings out the best in the American as well as the host country sub-

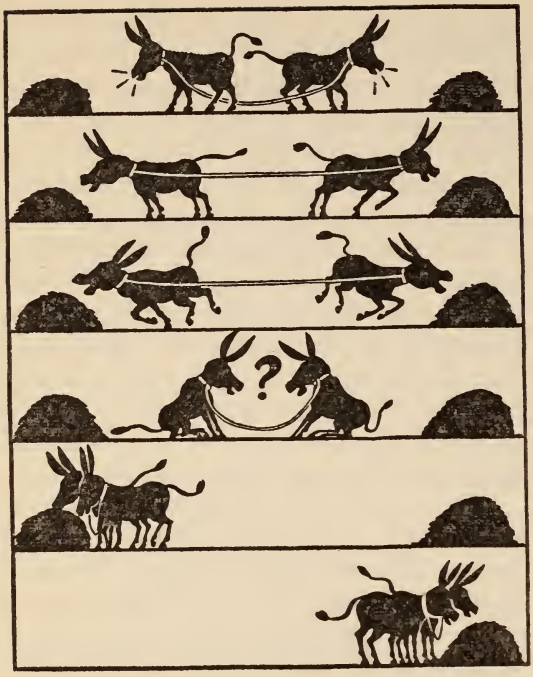


Fig. 3

ordinates. This day-to-day relation merits emphasis in that it often leads to long-term progress and long-term fruitful relations between professional personnel. Under these circumstances the American and his host country colleagues share office space, are subjected to the same day-by-day office routines, and work together on a variety of routine matters. It is often described as a shoulder-to-shoulder or shirt-sleeve relationship which does much to engender mutual respect and confidence. It provides innumerable ways to demonstrate, employ logic, and utilize experience in the daily round of business.

This environment provides the dignified and confidential atmosphere to apply suggestion, to employ criticism and, by the process of repetition of the minor or less conscious ways of attacking problems, provides the place for the host country technician to advance his understanding. This relationship is, of course, enhanced as the American improves his knowledge of the local language, customs and culture.

The fact that activities can only be performed through joint agreement and joint use of funds provides a tremendous catalytic force for action. The process of decision making raises the value of the activity in the eyes of the U. S. technician, the host country counterpart as well as the community recipient. It provides the sense of wholeness necessary to experiment with and adapt new procedures, methods and equipment to local circumstances. (Figure 3.)

A traditional pattern for operating rural public health units in underdeveloped countries is to line up the women and children, give each a number and then attempt to give help and consultation, taking one after another. At the end of a day there is usually still a long line of unhappy sick and huddled-up persons who have to return empty handed to their homes, which sometimes are miles away.

On hundreds of occasions, I have had the opportunity to work within these rural units and, after securing the understanding and and confidence of the local doctor, to work out with him ways to establish special clinics for mothers bringing babies with feeding problems, other scheduled clinics for the sick pre-school child, the well baby, the school child, the adult. The end result of such efforts is to increase the clinic capacity to handle many more problems. A variety of local by-products come from this, such as working with groups, the natural opportunity to introduce the preventive approach and the development of a positive health consciousness among the people. Many of our overseas personnel are intimately engaged in these important day-to-day types of activities.

I do not wish to leave the impression with you that all one needs to do is to find a way to physically get to these areas in the world and conditions will be improved and efficiency can be established. Aside from the problems of pulling up stakes and getting cleared for overseas work with the Government, a variety of obstacles beset us Americans when we attempt to grasp the meaning and significance of the cooperative program philosophy.

We find it difficult to share responsibility.

We tend to be impatient and suspicious, thus preferring a completely U. S. controlled operation to a joint operation. We tend to pride ourselves on a super-sophisticated technical and administrative approach to the solution of health problems. We tend to delegate as little as we can get away with and we shy from trusting our colleagues to carry responsibility in the way we would do. A very natural quality of Americans, or of anyone for that matter, is to assume as important a role as possible in any undertaking. The title we often give of advisor or consultant sounds very impressive and important. Many of us have been told the painful truth by our foreign colleagues, that it often takes one or two years for them to make a consultant out of an American.

We tend to underestimate people of another culture because of their paucity of material things. We tend to feel that a thing is done wrong if it is not done in a way we would do it. On the other hand, if we have pulled over our shoulders the cloak of a consultant, we are apt to overestimate people of another culture because of its reflected effects.

All of these considerations, in the last analysis, make us human. We are not born with ready-made good qualities and it is obvious that many things that make for success have to be learned on the job. Even the flair for teamwork in our culture may have quite a different meaning in a foreign country. There is no finishing school for this type of undertaking.

Many Americans view our activities as an exportation of know-how. Such a concept is not very acceptable to our foreign colleagues. We can characterize this process in a more acceptable manner by bringing out its main feature, which is adaptation—not copy-cat adoption—of known and successful methods to a local scene. It is this adaptation concept which provides an acceptable position enabling us to join with our foreign colleagues on equal ground.

Many American professional personnel have been abroad for years engaged in the cooperative development of health centers, special clinics, small hospitals, water systems and other health facilities. We can assure you that due to the adaptive process these health centers, clinics and other facilities have only a superficial resemblance to those in this country. If these facilities were imported back into the U. S. and compared to those here we would disclaim any relationship. The important point is that they fill a need and meet the requirements of local circumstances. Furthermore, they are operated by local personnel.

Thus we come to the final point in this section. During the past sixteen years, we have begun to see the growth of health institutions in many of the underdeveloped countries. By this we do not mean the bricks and mortar type. We mean the social institutions, like a profession of nursing or sanitary engineering. Our Public Health Service, for example, is an institution in this country and we have seen Health Services, Organizations, Departments, etc., appearing as strong and viable institutions in the underdeveloped and newly emerging countries. Schools of nursing, medicine, and other educational facilities have been created in many areas through technical cooperation.

Those of us who have had the opportunity to work through an entire program to the point where it goes under its own steam can point to joint achievements which are new under the sun. In short, there is a real creative characteristic to this work. Because of this creative quality we can all join wholeheartedly with our foreign colleagues in being proud of that which has been created through the process of technical cooperation. There has been a real ferment at work; we can point with pride to evidence of the development of human resources.

A WORD ABOUT THE FUTURE

Many of you have heard of the world-wide project to eradicate malaria. Such a bold idea had its origin in our U. S. experience in eradicating malaria within our borders. The

Fig. 4

same results have been achieved in several other countries: Italy, Sardinia, Chile, Argentina, Mauritius. The 88 members of the World Health Organization agreed that such an objective on a world basis was feasible. Such a vast project could not be carried out without the participation of our Government and it was Dr. John Hanlon who took the first step in ICA to start the wheels of program planning turning until a year ago next month we formally joined in this great human and professional adventure (Figure 4).

Today we can point to extensive experience in other fields such as the control of the diarrheas and dysenteries through development of potable water, the control of trachoma, tuberculosis, schistosomiasis and other diseases. Those of you who have traveled abroad know what I am talking about when I mention the health hazards caused by precarious water and food sanitation in foreign countries, especially in the lesser developed nations. In many large areas of the earth, safe clean water, just to satisfy normal physiological requirements, is a scarce commodity.

Recently Mr. Robert W. Kitchen, our Mission Director in Sudan, reported on a study made in a large area near Khartoum. They found that water is so difficult to get that the average family of six allocates the full time efforts of two members to the business of finding and bringing home sufficient water for daily needs.

In many places in South America it is not uncommon for a family to spend between one-tenth to one-fourth of its total income for water, usually dirty and unsafe and the cause of much illness, disability and death.

If one compares the relatively small cost of potable water in our society with the heavy burden it represents to millions of families in the lesser developed countries, one is impressed at the high cost of disease, ignorance and poverty.

We in ICA are attempting to analyze our 16 years of experience in this important public health sector and hope to make an all out effort to help millions develop national community water supply programs to spearhead an attack on the leading health problem in the world's less developed area.

Our future will involve special attacks on these and other basic problems as fast as we can make the necessary professional, administrative and financial feasibility studies. In my opinion, no greater challenge can be found in our profession than that presented by these world health problems. No greater

compensations can be gained today than to participate in the solution of many of these problems and to see the fruits of one's own efforts in healthy productive people.

Many of our Statesmen who have struggled with the great problems of international collaboration have recognized that technical cooperation leads to the development of healthy, productive and independent people in place of the sick, miserable and dependent populations.

President Eisenhower in his State of the Union speech of this year, and in his recent address before the United Nations, emphasized the importance of health in the fabric of peace. He has indicated that the U. S. A. will always go the last mile with any nation wishing to collaborate in health. We have a great challenge before us. I believe we are ready and able, that we can and will continue to accept this challenge.

W. W. Keen's "First Modern Brain Case"[1]

By DONALD E. WILLARD, JR., M.D.[2]

DR. SCHEFFEY, Dr. Walker, Ladies and Gentlemen: Infancy usually appeals to us. The abiding peril, unforeseen by a baby, arouses our sympathy. While at the same time, the wonderful possibilities for its future excite our imagination. Later on, the future arrives; the weaknesses have become strengths, the possibilities, realities. Sometimes we look back with great interest to the child that used to be.

My pleasant task this evening is to recount for you certain events of the nineteenth century during the infancy of American neurological surgery. I would like to discuss particularly some of the contributions of a great American surgeon—Dr. William W. Keen. With his first attempt at elective surgery within the brain, he successfully removed a large meningioma. The patient went on to live for thirty years as the first person in America to be cured of a brain tumor.

To begin, I want to relate something of Keen's own medical career; and, also, his reactions to the important discoveries of medical science which had to precede his "first modern brain case." After that, I shall discuss the case itself in some detail.

W. W. Keen began his medical career by entering the Jefferson Medical College in 1860. At the end of the first year he was drafted for service in the Union Army (1). However, he managed to return to Jefferson in time to graduate with his class in 1862 (2). Immediately after graduation, he again joined the Union Army. But this time, instead of caring for casualties at the front line, he was ordered to serve at the Turner's Lane Hospital in Philadelphia under Weir Mitchell (3). Keen said later that this work with Mitchell "was the chief turning point of my whole professional life" (4). They developed a close friendship which was to last for over fifty years. But equally as important, they undertook a long-term study of nerve injuries in wounded soldiers. This work led to a number of publications the most famous of which was *Gunshot Wounds and Other Injuries of Nerves* (5).

In 1921 Dr. William H. Welch said that this early work by Keen and his associates "did for the peripheral nervous system what Duchenne and Charcot had achieved for the spinal cord" (6). Sixty-five years after the Civil War Keen himself said, "Our war work laid the foundations of modern neurological surgery" (7).

However, even though he had this distinct, early interest in neurological diseases, his first effort in elective brain surgery had to await two very necessary advances in medical science. The first of these prerequisites was embodied in what is perhaps the most significant paper in all surgical literature—"On the Antiseptic Principle and the Practice of Surgery" by Joseph Lister (8). In 1876 Lister came to Philadelphia to present his thesis. Keen heard him speak and immediately accepted Lister's ideas and methods. He was the first Philadelphia surgeon to employ the antiseptic technique (9, 10).

Prior to this time, the danger of infection was so great that all operative treatment stopped at the dura. During the Civil War, the mortality from trephining alone in cases of fractured skull was over sixty per cent (11).

The second important discovery was made by Fritsch and Hitzig and was enlarged by the work of Ferrier, Horsley and others. These men found that, in contradiction to existing concepts, the brain does not function as a homogeneous tissue like the liver. Instead,

[1] Edward Bell Krumbhaar Prize Essay II, Section on Medical History, College of Physicians of Philadelphia, 21 October 1958.

[2] Intern in Surgery, The New York Hospital, New York 21, N. Y.

the many functions of the brain are parcelled-out to specific cells in specific locations.

Keen was well informed of this work also. In 1885 he made this statement: "As a result of their labors, the brain is now mapped out with reasonable accuracy so that, given hitherto ill-understood or obscure localized symptoms we can now say that there is certainly a tumor, an abscess or other disease in precisely this or that locality" (12). Until this concept was established, no basis existed for assuming that clinical symptoms and signs could be used to predict the site of a focal lesion in the brain.

Time passed. By 1887 Keen was fifty years old. His surgical practice was large; and, in addition to these duties, he was professor of surgery at the Woman's Medical College. He also held several other positions at this time. He was professor of anatomy at the Pennsylvania Academy of Fine Arts and a Trustee of Brown University.

Nevertheless, in November 1887 he went to New York to watch his old friend Robert Weir remove a brain tumor (13). This lesion had been well localized clinically. Weir followed the general procedure that had been outlined by Horsley (14). However, this was Weir's seventh case of brain surgery so he was in a position to instruct Keen on the basis of some personal experience.

A month later the Philadelphia surgeon operated upon a patient himself (15–18).

The patient was a twenty-six year old man from Lancaster, Pennsylvania. He was first seen by Keen at the St. Mary's Hospital in Philadelphia on May 30, 1887.

The history, as pieced together by Keen, began when the patient was three years old. At that time he had fallen from a window and had struck his head on some bricks. His scalp had not been cut, but his forehead was indented on the left side. He was comatose for about an hour. However, he recovered from the injury with no apparent after-effects.

Then, at the age of five, the man had an attack of measles which was complicated by a right ear infection. A chronic discharge developed which continued intermittently up to the time of admission.

Throughout his boyhood, the patient was considered dull and very forgetful. He suffered from frequent headaches. He continued, otherwise, to have good health until the autumn of 1884. At this time he developed severe, generalized neuralgic pains which gradually became worse. In February 1885 he was seized with violent epileptic attacks followed by intense head pain. By the end of April 1885 he was hemiplegic on the right side, and had a right hypalgesia. In June, when he was seen by a Lancaster physician, he was unable to talk. At this time a small scar was discovered on the left side of his scalp. Pressure on this scar increased the head pain.

The Lancaster physician diagnosed a space-taking lesion of the anterior lobe of the left hemisphere involving the third convolution and extending backward. He treated the man with iodide of potassium and arsenic with laxatives.

With this therapy the patient improved remarkably. Gradually, all of his symptoms disappeared. But in their place he began to lose his eyesight. Within a few months he was totally blind; by now the original symptoms were beginning to return also.

Thus, when he saw Keen in May 1887, he was in no mood to quibble about his chances of surviving an operation. He earnestly desired surgical treatment if it promised even the slightest chance of some relief.

Keen, on the other hand, was cautious. After all, the man had improved on a therapeutic regimen known to cure syphilitic gummas of the brain. In addition to his own examination, he had the patient examined by three neurologists including Weir Mitchell and an ophthalmologist.

The consultants made very careful studies and came to a conclusion which confirmed Keen's doubts. They thought the patient had a growth in the left motor area causing the irritative phenomena and a destructive basilar lesion causing the eye symptoms. They reasoned that a syphilitic gumma in the left

hemisphere with a subsequent basilar meningitis could produce such a clinical picture. The man was again treated intensively for syphilis. No effect whatsoever was noted after five months.

Still very miserable, the patient came back to St. Mary's in December 1887. This time everyone decided that the growth was not a gumma but more likely a tumor of traumatic origin. This was not an unusual diagnosis (19). Actually, nearly all the textbooks of neurology in that day referred to accidental tumors or tumors of traumatic origin as a distinct brain disease.

The St. Mary's Hospital of 1887 had previously been a private home and had very primitive facilities. But very careful preoperative preparations improved these. In the patient's room all unnecessary furniture and the rugs were removed. The walls and ceiling were carefully cleaned a day in advance and the wood-work, furniture and floors were scrubbed for several hours preceding the operation.

All of the instruments to be used were boiled in water for two hours. Brand new deep-sea sponges which had been kept in a strong carbolic solution were used. These sponges look to me like large pancakes but in those days they were called "elephant ears" (20).

The patient's head was shaved the day before, scrubbed with soap and water, then with ether, and finally, covered with wet sublimate dressings. When the operation began, the dressings were removed and the head was scrubbed again.

Those participating in the operation scrubbed their hands and nails thoroughly with soap and water, then rinsed in alcohol and sublimate solutions. This was antisepsis, not asepsis. Sterile gowns were not used; rubber gloves, unheard of.

When the operation finally began, the time was one P.M. In addition to Keen those present included eight attending physicians, the resident house staff and two medical students.

Confident that a tumor of traumatic origin had arisen from the site of injury as indicated by the scar, Keen's first step was to mark the position of the scar on the skull. The scar was incised down to the bone and a little nick was made in the skull with a gouge. He then reflected a large semicircular flap of scalp. Bleeding was controlled by hemostats.

Next, a hand trephine one and one-half inches in diameter was used to remove a button of bone which included the nick representing the scar. A second button was removed just posterior to the first. This done Keen palpated through the dura and felt a hard mass still extending beyond the limits of the skull opening. Such luck is amazing to me

The opening was further enlarged by snipping away the bony margin with rongeur forceps. When this was completed, a skull defect measuring two and one-half by three inches was present. At this point Dr. Keen reports, "My heart sank down into my boots But dangerous as the procedure might be, I had to go ahead" (21).

Going ahead, then, he found on incising the dura that it was adherent to the tumor This being the case, he cut the dura around the entire circumference of the exposed area Gently, he introduced his little finger in behind the tumor and with very little force shelled it out of its resting place.

The enucleation tore several large veins and these bled freely. With great difficulty he succeeded in ligating them. The hemorrhage continued. He douched the cavity with hot water and applied pressure with the "elephant ears." After about twenty minutes, the bleeding stopped.

Two rubber drains were inserted into the cavity and a bundle of horsehairs was laid across the wound. Although he had preserved the bone fragments with the hope of replacing them, his removal of the dura prevented this. He merely closed the scalp wound and applied a thick dressing consisting of two sponges, sublimate gauze, rubber dam and a muslin bandage topped off with a skull cap.

The entire procedure lasted about two hours.

The patient's post-operative course was stormy. During the first week, a large clot

formed beneath the skin flap. This hemolyzed and had to be washed out by gentle pressure and douching with sublimate solution. The man's preoperative symptoms subsequently began to diminish. Late in the second week the symptoms returned. The skin flap bulged considerably; the patient's temperature rose to 104°F. and he developed diarrhea. Keen feared an infection. He reopened the wound with his finger and found, instead of pus, a large friable mass of brain tissue. A hernia cerebri pushed its way through the gaping incision. Keen pardoned himself, later, for this error on the basis of inexperience.

By the fourth week the neurological symptoms had subsided considerably but the hernia had gotten larger. In addition a little pus and a great deal of spinal fluid had begun to ooze from the granulating surface. Finally, the discharge ceased but the wound did not heal. So Keen resorted to skin grafts which were taken from the patient's arms and placed directly over the surface of the hernia be tween the wound edges.

Gradually the incision healed and the hernia subsided. Suddenly, on the seventy-fifth day, the bulge was absent and in its place was a deep hollow pit two inches deep. On the eighty-fourth hospital day the patient was discharged from the hospital.

Keen realized that with such a defect the man's brain had to be protected. So he bent a piece of tin to fit the contour of the skull, covered the tin with silk, and sewed this to the inside of a skull-cap. The patient wore this protective cap for the rest of his life.

Finally, in 1918 the patient died. Keen was by this time eighty years old, his large experience in brain surgery belonged to an earlier era. But he never lost interest in his first case. Upon hearing of the man's death he sent a pathologist to Lancaster to procure the brain. On examination not only was the large cavity present but the bottom of it was formed by the floor of the fourth ventricle. For those of you who may be interested in it, I brought that very same brain with me tonight. It was loaned to me by Dr. David R. Morgan, Professor of Pathology and Curator of the Museum at Jefferson Medical College.

In conclusion, I quote a comment by Dr. Harvey Cushing, who was a freshman at Yale in 1887:

> I would like to . . . doff my cap to the soul survivor of (the) pioneers in this work. My fellow countryman W. W. Keen who was one of the very first to localize and remove successfully a large meningeal tumor yet had neither bone wax to use, nor muscle to implant or hypertonic saline at his command. No knowledge of intrapharyngeal narcosis, far less of local anesthesia, no instrument of precision with which to follow the patient's condition from moment to moment, no provision for transfusion in case of need and who simply made a sufficiently large opening in the skull, removed the exposed tumor with his finger, ligated the bleeding vessels, drained the wound and in spite of a subsequent fungus cerebri, the patient with the same help that Paré relied upon, ultimately recovered. Both Dr. Keen and his celebrated case, the end result of which he recorded thirty years later, are exceptions to all rules, neither is likely to be duplicated (22).

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Second Edward Bell Krumbhaar Award in Medical History

IN APPRECIATION of his outstanding contributions to medical history, the Section on Medical History of the College of Physicians of Philadelphia, in 1957, named its medical student essay contest for Dr. Edward B. Krumbhaar, past president of the College and emeritus professor of pathology at the University of Pennsylvania. The first winner of this award was Dr. Marshall Smith, graduate of the University of Pennsylvania now a resident in medicine at the Presbyterian Hospital of Philadelphia.

The second Edward Bell Krumbhaar Award was presented to Dr. Donald E. Willard, Jr., 1958 graduate of Jefferson Medical College now surgical intern at The New York Hospital, on October 21, 1958. His essay, "W. W. Keen's 'First Modern Brain Case' ", was judged the best of six entries for 1958 by a committee consisting of Drs. Arthur M. Dannenberg, Herbert J. Dietrich, Jr., and Samuel X Radbill. The Award of one hundred dollars and a prize certificate was presented to Dr. Willard, on behalf of the Section on Medical History, by Dr. Krumbhaar. (The prize essay will be published in the *Transactions*

Left to right: Dr. Jonathan E. Rhoads, President of the College of Physicians of Philadelphia, Dr. Edward B. Krumbhaar, Dr. Donald E. Willard, Jr. (Prize essayist), Dr. Fred B. Rogers, Chairman of the Section on Medical History.

& *Studies of the College of Physicians of Philadelphia* for February, 1959). This paper was read by Dr. Willard at the College Hall on March 11, 1958, at a meeting co-sponsored by the Osler Medical History Club of Jefferson Medical College. Dr. David J. LaFia is faculty advisor to the Jefferson group.

A dinner was held at the Hall of the College prior to the prize award for student essayists who submitted papers during the past year. Dr. Jonathan E. Rhoads, president of the College, and Dr. Lewis C. Scheffey, immediate past president, joined Section members and friends on this evening.

The Section's annual medical history essay contest, open to all students in Philadelphia's five medical schools, is now inviting entries for its third Edward Bell Krumbhaar Award, to be awarded late in 1959.

HERBERT J. DIETRICH, JR., *Clerk, Section on Medical History*

Memoir of John Allan Bertolet (1889–1956)*

By LEWIS C. SCHEFFEY, M.D., SC.D. (HON.)

IN THE course of securing a medical education in these days, it would not be unusual for a lad of seven to witness his father's graduation from medical school. In 1896, however, such an occasion was indeed rare, but the subject of this memoir did just that. Twenty years later the situation was reversed, and John Allan Bertolet received his M.D. degree on the Academy of Music stage in the time-honored tradition of Jefferson Medical College commencements, his distinguished parents Dr. John Marshall Bertolet and Louisa Deeter Bertolet being in the audience.

The Bertolet family was originally of French extraction; the spelling then was "de Bertholet". Auguste Bertholet, a distinguished officer in the French army was killed in the course of the allied victory at Yorktown in 1781. Dr. Bertolet, however, was a descendant in the seventh generation of the American line that stemmed from Jean Bertolet, a Huguenot who fled Alsace with his family by way of Switzerland, and finally settled in the Oley Valley, southeast of Reading, Pennsylvania in 1726. The homesteads of Mordecai Lincoln and George Boone, forefathers of the martyred President, and Daniel, the exploring huntsman, also lie in the immediate vicinity.

Born in Reading, Pennsylvania, May 26, 1889, Allan, as he was usually called, graduated from Reading High School in 1907, from Phillips Exeter Academy in 1908, and from Princeton University in 1912. When, during a summer holiday, he worked his way to Europe as a stevedore on a freighter, one might readily glean that he was strikingly athletic in appearance from the manner in which the Mayor of Reading referred to him in the following letter:

June 15, 1910

To Whom It May Concern:

J. Allan Bertolet is a son of Dr. John M. Bertolet of the City of Reading, State of Pennsylvania, U. S. A. The young man is 21 years of age, 6′2⅛″, 190 pounds in weight, has dark eyes, light complexion and is muscular. He is taking a trip through Europe, and whatever courtesies can be extended to him will be appreciated.

Yours truly,
William Rick,
Mayor

He was in truth an all-around athlete, excelling in track and field sports and in football. Four years his junior throughout our school days and thereafter, he inevitably became my boyhood hero, and as the years passed, a firm and sincere friendship continued throughout our respective lives.

Dr. Bertolet's internship at Jefferson Hospital was interrupted by enlistment in the Army Medical Corps, and an assignment with U. S. Base Hospital № 38, going overseas June 5, 1918 and completing two years service as Captain in this Jefferson Medical College Unit. His record was a distinguished one, and he was promptly accepted at Jefferson after the war ended for residency work in general surgery and otolaryngology during 1919–1920. Hospital appointments later included a service in otolaryngology at the Protestant Episcopal Hospital of Philadelphia, and an assistantship in the same speciality at Jefferson. Likewise he held a teaching position as Associate in Otolaryngology at the Graduate School of Medicine, University of Pennsylvania. For a period of ten years he served as

* Prepared and published at the request of the Council of the College of Physicians of Philadelphia.

consultant in a similar capacity to the Medical Department of the Pennsylvania Railroad.

A Fellow of the College of Physicians of Philadelphia, having been elected in 1937, he also enjoyed membership in the American Academy of Otolaryngology, and in the Philadelphia Laryngological Society. He was a Fellow of the American College of Surgeons, American Medical Association and Medical Society of the Commonwealth of Pennsylvania. At one time he was secretary of the Aesculapian Club of Philadelphia and held membership in Benjamin Franklin Post 405 of the American Legion.

Dr. Bertolet took an active part in organized medicine, serving as a Director of the Philadelphia County Medical Society from 1938 to 1941, and was elected Treasurer of the Society from 1942 until 1949. With the death of Dr. Herbert Goddard in 1944, the office of Coroner of Philadelphia became vacant, and Governor Edward Martin appointed him to fill the unexpired term. Then in 1945 he was nominated and elected to the regular 4-year term on the Republican ticket, an office that he filled creditably. It was at this time that Dr. Bertolet's splendid physique began to fail, invalidism gradually forcing his retirement from public office and from the practice of medicine as well. It was with sorrow and disappointment that he accepted the inevitable, passing from this life on April 6, 1956.

Allan Bertolet enjoyed the work of his profession. He practiced excellent medicine, and operated skillfully. He liked people and people liked him. As a public official and in his political relationships he comported himself with dignity, assurance and scrupulous honesty. His many friends can attest to his nobility of character and courage in the face of long and protracted periods of illness.

His personal and family life was a happy one. On December 1, 1920, he married the former Helen W. Wilbee of Plainfield, New Jersey, who survives him. Mrs. Bertolet was a constant companion of her husband, faithful in her attachment and ever solicitous for his comfort and well-being. She truly enjoys the sympathy, respect and love of all the friends and colleagues of Allan Bertolet and herself.

Memoir of Everett Pusey Barnard (1875–1957)*

By PHILIP F. WILLIAMS, M.D.

DR. BARNARD, who became a Fellow of our College in 1911, died on November 2, 1957. He was born on March 1, 1875 on the family farm in Lancaster County where his ancestors had settled after emigrating from England in the late 1600s.

After early education in a one-room red schoolhouse nearby, he attended Wyoming Academy at Kingston, Pennsylvania. At the time his class graduated he refused to speak the customary oration required of each pupil at the commencement exercises and was refused a diploma. However, on the 25th anniversary of graduation the faculty relented and sent him the diploma.

He graduated from the Medical School of the University of Pennsylvania in 1900. While at the University he helped organize and played on the first basket-ball team. He never lost interest in this phase of University athletics and seldom missed the annual home game with Princeton. On the Varsity foot-ball team of 1899 he was one of the full-backs and an excellent punter.

In the early years of practise he was closely associated with John G. Clark, as anesthetist, and with Richard C. Norris, as assistant at the Preston Retreat. At their suggestion he went to the famous Rotunda Hospital in Dublin, and to the Royal Infirmary, Edinburgh, for graduate work in obstetrics. After returning to Philadelphia he joined the teaching staff at the University, and was appointed to the Preston Retreat, Maternity Hospital, Germantown Hospital and Lankenau Hospital. Dr. Barnard was deeply interested in the Lankenau Hospital, and was frank in his dislike of the removal of the institution to the suburban location it now occupies. His clinical teaching in the latter two institutions was highly regarded by a generation of internes and residents. He was elected a Fellow of the College of Physicians of Philadelphia on November 1, 1911.

His many years of busy practice were interspersed in appropriate seasons with fishing expeditions to the trout streams of Northern Pennsylvania, with upland hunting for game birds and small game, and duck shooting over the Delaware and Susquehanna flats. An excellent swimmer, he early gave up that form of exercise. During a summer vacation while in medical school he acted as a life guard at a New Jersey beach. While rescuing a stout female bather who had ventured beyond a safe depth he nearly lost his own life. This heroism ended his aquatic career. His native hardiness, accentuated by his athletic activities, was probably responsible for his habit of not wearing an overcoat, even in midwinter.

A low handicap golfer and at one time champion of the Philadelphia Country Club he won many trophies. The one of which he was especially proud was a small silver trophy, a few inches high, inscribed "Lost to Duty". The story he told of it related to a "finals" match. He had his opponent three down and four to play when he was urgently summoned to follow the stork. The cup was a gift from the grateful patient.

Dr. Barnard, although voluble in private conversation had an aversion to public speaking, first demonstrated at Wyoming Seminary, as a decided characteristic. At one time he was asked if he would agree to be nominated for the presidency of the Obstetrical Society. He quickly refused on the grounds that he would then be forced to talk. Thereupon he was elected Curator (a non-speaking office) for some ten terms.

* Read before the College of Physicians of Philadelphia, 1 October 1958. Prepared and published at the request of the Council of the College of Physicians of Philadelphia.

A non-smoker and a teetotaler, he explained, was due to a boyhood promise to his Mother, a member of the Pusey family and of the Society of Friends.

His collection of antique and early American furniture was outstanding, and he knew the history of the makers and the styles. One of the pieces in his office was a beautiful mirror. On the back of it he noted that it had descended to him from a "Truman" ancestor, described the item, and ended up by writing "the 'Truman' mentioned above was no relation of the 33rd President of the United States"!

Dr. Barnard is survived by his wife, two sons, James T. and George B. and a daughter, Mrs. Henry Whittelsey.

Memoir of William Zentmayer (1864–1958)*

By BURTON CHANCE, M.D.

DOCTOR WILLIAM ZENTMAYER was born in Philadelphia October 28, 1864. He was one of five children of Joseph and Catherine (Bluim) Zentmayer. His father came from Germany and established himself in business as an instrument-maker in Philadelphia in 1853. He became eminent in his field, being interested in the construction of intricate instruments of precision. He was interested in scientific investigations and was the *first* to grind sphero-cylindrical lenses. He was awarded the Elliott Cresson gold medal by the Franklin Institute, as well as gold medals at the Centennial Exhibition in Philadelphia, 1876, and the Paris Exposition, 1878, for his work on microscopes. The microscope for which he was given the Centennial medal is now in the Mütter Museum of the College of Physicians of Philadelphia. He insisted that customers coming to him for spectacles should present an ophthalmologist's prescription before he would proceed to fill the order.

William completed his studies at the Medical School of the University of Pennsylvania, from which he was graduated M.D. in 1886.

Dr. Zentmayer began his clinical experience at Children's Hospital, in the Out-Patient Department, in 1887. In 1890 he became assistant surgeon at the Wills Hospital in the service of Dr. William Norris, whom he succeeded as attending surgeon in 1901; this service he continued until his resignation in 1928, becoming consulting surgeon from that time until his death. In the meantime he had been appointed surgeon at St. Mary's Hospital in 1893, serving there until his retirement in 1951.

From this active experience he found material for the numerous clinical reports published, many of which were delivered at the College of Physicians of Philadelphia. He was elected a Fellow of the College March 7, 1894 and remained a Fellow until his death.

In 1916–17 Dr. Zentmayer was chairman of the Section on Ophthalmology of the American Medical Association. In 1926–27 he served as President of the American Ophthalmological Society. He served as chairman of the Section on Diseases of the Eye, Ear, Nose and Throat of the State Medical Society in 1935–36.

During World War I he was in the Volunteer Medical Service Corps. In 1941 he was guest of honor at the annual meeting of the Section on Ophthalmology of the American Medical Association.

Dr. Zentmayer served on the editorial board of the *Archives of Ophthalmology* from 1950, when it was taken over by the American Medical Association, until the time of his death. He served as editor of the *Transactions of the College of Physicians of Philadelphia* from 1899 to 1913.

He was elected a Fellow of the American Ophthalmological Society in 1905, and a Fellow of the Ophthalmological Society of the United Kingdom in 1925.

He was abstemious in his personal habits, enjoyed music, for several years attending regularly the concerts of the Philadelphia Orchestra. He was unmarried. So thorough was he in the study of his own cases, and so ready in recalling references to the literature that Dr. Zentmayer was most helpful in consultation, in which he was generous, kind and patient. He was a great walker. While frail in appearance, he was remarkably vigorous. He was an avid reader with a remarkably retentive memory.

Dr. Zentmayer's technique as a surgeon won the admiration and approval of his assistants

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and the beholders. He was Professor of Ophthalmology at the Graduate School of Medicine of the University of Pennsylvania and his pupils were most anxious to possess copies of his lectures.

A striking portrait of him was painted by Daniel Garber and presented as a gift to the College of Physicians of Philadelphia by an international group of ophthalmologists, to show their appreciation of his devotion to ophthalmology.

In 1945 Dr. Zentmayer was awarded the Leslie Dana gold medal by the St. Louis Society of the Blind, "in recognition of his outstanding achievement in the prevention of blindness and conservation of vision"; and, in 1948, the Lucien Howe gold medal of the American Ophthalmological Society, "for his work in the domain of medical education and ophthalmology and for original investigations in his field."

He attended and took part in scientific meetings. He was a Fellow of the Academy of Natural Sciences of Philadelphia, and a member of the Pennsylvania Academy of the Fine Arts, the Medical Alumni Society of the University of Pennsylvania, and the University Club.

Dr. Zentmayer died March 18, 1958, following a brief illness, and was buried in Westminster Cemetery, Bala-Cynwyd, April 3, 1958.

From his private fortune he bequeathed to the College of Physicians of Philadelphia $15,000 for the establishment of the William Zentmayer Book Fund; $1,000 for the use of its Section on Ophthalmology; and, after the disposal of certain other funds, one-ninth of the residue of his estate. These bequests will be distributed following the death of his sole surviving sister, with whom he lived.

Memoir of Williams Biddle Cadwalader (1876–1957)*

By GEORGE W. NORRIS, M.D.

PRESIDENT RHOADS: Fellows of the College; Ladies and Gentlemen: Many years have elapsed—about forty I believe, since I was last asked to speak in this hall. That I should be asked to do so again and in so doing be given the opportunity to reminisce about one of my oldest, most intimate and beloved friends is a high honor and one that I greatly appreciate. Frankly, it touches me deeply.

This audience will understand, I hope, that when in my discourse I refer to Doctor Cadwalader as "Bill", it denotes no lack of respect on my part. On the contrary, it is the natural, the instinctive, the affectionate appellation used in referring to an intimate, life-long friend. It comes from the heart!

Williams Biddle Cadwalader—named for an ancestor, Jonathan Williams, whom George Washington appointed as the first Commandant of the West Point Military Academy—was often greeted by his host of friends as "Bill". He and I had been intimate friends since the eighteen-eighties, when we lived less than half a block apart on Locust Street near Sixteenth. Those were the days when sidewalks were of brick or flagstone, with spotlessly clean, white marble steps leading to the front door, before each house. At night both houses and streets were feebly illuminated by gas, and all shutters were bolted to discourage intruders. But for the occasional clap-clap of horses hoofs, silence reigned supreme. By day we had music furnished by an Italian organ-grinder, whose monkey scaled the water spouts for pennies, to the delight of the children, or by German bands to whom one tossed pittances and often waved them on.

We lived our early youth in the mid-Victorian era, during which many fine old colonial houses were demolished and replaced by hideous, gloomy, architectural monstrosities. This era was one in which ultra-nicety and hypocrisy prevailed. When dining-out, it was considered bad taste to express preference for a chicken's "thigh"—"second joint" was a more polite term. In those days the gossips whispered: "I hear that his house is mortgaged", or "I understand that she is contemplating a divorce". At that time streetcars were dragged by emaciated horses, whose span of life, after being thus employed was about eight years! Fire engines drawn by galloping horses raced down the streets paved with cobblestones or Belgian blocks. It was customary to sprinkle tan bark, to a depth of six or more inches, on the streets, to muffle the hub-bub of traffic in front of the residence of a person of prominence who lay gravely ill.

Yes indeed! Customs have changed a lot since Bill and I were young—perhaps not for the worse, but profoundly. But "The moving finger writes, and having writ, moves on"!

Of course we were a part of this Victorian Age that surrounded us. Possibly there was something in the very air of the times that brought us together; but, close we were and close we remained through life!

After preliminary schooling at St. Paul's in Concord, New Hampshire, and later Princeton College, Bill obtained his medical degree from the University of Pennsylvania in 1902. At the suggestion of a relative, Dr. S. Weir Mitchell, he decided to specialize in Neurology. He did so by working for a number of years in the laboratory of Professor William G. Spiller, and there became well-grounded in the anatomy, physiology and pathology of his chosen subject. Later, he often spoke most

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appreciatively of the kindness and profound knowledge of his preceptor. Years afterward, he himself became Professor of Neurology, and was President of the American Neurological Society in 1939. The honorary degree of Doctor of Science was conferred upon him by the University of Pennsylvania in 1952.

Bill was a loyal citizen, and like many others of the Cadwalader family, served his country in time of war. In the Spanish-American War, he was a member of the First City Troop. During World War I, he served in France with U. S. Base Hospital No. 10 (the Pennsylvania Hospital Unit). He was detached from this organization to act as Neurological Consultant in the American Army, with the rank of Major. As such, he was called upon to see many cases such as we had encountered while serving with the British at Le Treport, where men arrived from the front tagged: "N.Y.D. Shell shock, D.A.H." (Not yet diagnosed, shell shock, disordered action of the heart). In London, we had visited a special hospital, staffed by expert cardiologists, internists and neurologists at which "D.A.H." was being studied. It was precisely the same condition as that which had been described during our Civil War by Dr. Jacob M. DaCosta—an erstwhile president of the College, whose portrait still adorns our walls—as "Soldier's Heart"!

Bill was broad-minded, scholarly, widely read and well informed. He could talk on any subject intelligently and interestingly. He was a man of diverse interests and hobbies. He was a sportsman. He habitually cast flies for trout in the Poconos, and had fished in the waters of England, Austria, Hungary and even Iceland. He also spent many spare hours in shooting quail and pheasants. Like everything else in his career—what he did, he did well.

Bill Cadwalader was associated with many organizations such as the Society of Colonial Wars, the Delta Psi Fraternity, the Philadelphia Club, the Academy of Natural Sciences, the Association for Research in Nervous and Mental Diseases, the American Committee for Wildlife Preservation, the American Society of Mammologists, etc. For many years he was associated with the Mutual Assurance Company as a Trustee, and the "Medical Club of 19" as a beloved friend. His services to the City of Philadelphia anent the Zoological Society cannot briefly be set forth; nor over-rated by length! A step in doing him justice has already been taken by this Society in the publication of a memorial portrait brochure.

It was through Bill's initiative, and Dr. Charles Penrose's generosity, that the Pathological Laboratory, under the egis of that well-known pathologist, Doctor Herbert Fox, was established. This institution was the first of it's kind in this country. The studies carried out on animals, birds and reptiles, led to better methods of feeding, health and longevity.

Among the early results was the discovery that monkeys could be allowed out of doors in winter, of their own initiative, without detriment. The establishment of a period of quarantine for all new purchases was most beneficial. (Putting them in glass cages where tuberculous humans could not spit at them eliminates the most frequent cause of the monkey's death). When horse meat and some other special foods became almost unobtainable, formulas were worked out at the Laboratory, containing adequate amounts of carbohydrates, proteins, fats, roughage and vitamins, which even the carnivora could gradually be trained to eat with gusto!

For many years, a large part of Bill's thought, time and energy was devoted to the Zoological Society with which he was associated from 1890 to the time of his death. On it's behalf he visited zoos abroad to observe methods and worthwhile innovations. Thus, with the loyal cooperation of the rest of the Board, the Fairmount Park Commission and the City Authorities, the Zoo not only survived the financial depression, but was rebuilt, refurbished and today ranks with the best in the world! This has been ably and accurately set forth in a letter from the late lamented Charles I. Thompson, Esq., for many years President

of the Fairmount Commission, from which I quote:

"Observing that the Park Commission had obtained substantial funds from the City for the construction of an Art Museum, he decided to make a similar effort on behalf of the Zoo. As his first step he sought to procure an appropriation of $1,000,000 for new houses for animals and birds.

"You must bear in mind that there is a pork barrel aspect to the allocation by City Council of funds for improvements. Each of the City Fathers likes to sponsor a project which will please his particular constituents and get him votes. Animals can't vote and voters do not live in Fairmount Park. Living in Montgomery County and never having been active in politics, he had literally nothing to sell except his own charm and personality.

"As his first step in building a friendly relationship with the members of Council, he began inviting the members to attend a spring party at the Zoo followed by a convivial dinner at the Rabbit.

"Dr. Cadwalader's preliminary wheedling of City Council was, of course, not his primary Zoo activity. He lunched at the Garden twice a week and spent the afternoons wandering from building to building inspecting the structures and getting to know the animals by name. When a new exhibit arrived he was always present.

"During the latter part of the depression years of the 30s and throughout the War, the good Doctor made a point of getting to know each and every Councilman by name. He pounded the corridors of City Hall and visited each and every Councilman in his office. He sought to inspire each with his vision of the future of the Zoo and by his enthusiasm and perseverance gradually won each over to his point of view.

"In 1945 Council unanimously authorized a Councilmanic loan of $1,000,000 for Zoo purposes. In the 30s when his campaign had started, this sum would have sufficed for reconstruction of a major portion of the Zoo. Inflation and high prices brought on by the War, cut down the effectiveness of the grant and it was found that it would only suffice for a magnificant new Carnivora House. He presided at the dedication of this structure in 1951.

"Undeterred by the inadequacy of the appropriation to accomplish his full purpose, he was then instrumental in obtaining additional funds from generous donors for the new Elephant House, Bird House and, shortly before his death, the Children's Zoo.

"Some years ago he established the policy of personally presenting gold watches to employees with twenty-five years of service. This became an annual occasion, and following each spring board meeting, the employees with the requisite service were called in. Dr. Cadwalader would call each by his first name and present him with his watch. The board turned the tables on their President in 1951 after he had served twenty-five years in that capacity. On that occasion they presented him with a silver cigarette case inscribed by each member.

"It was also Dr. Cadwalader who conceived and started the annual members' parties at the Zoo which have been held each spring since 1950.

"It is, of course, no exaggeration to say that Dr. Cadwalader knew every employee intimately and by his first name. It is also true that he knew each of the animals by name. As an example of his familiarity with each, my secretary tells the following story: She and her sister were visiting the Zoo about three years ago. They came near the moat enclosure and found a charming elderly gentleman inside the enclosure holding a one-sided conversation with Frankie, the hippopotamus. After a few fond embraces and an affectionate hug, he departed. Later they saw the same gentleman holding another conversation with the shoe-bill stork. This time he said his goodbyes by picking up the stork and kissing it fondly on the bill. She later recognized the man as Dr. Cadwalader.

"In 1955 Dr. Cadwalader made a personal plea to the City Administrative Officials, the Planning Commission and City Council for planning money for the completion of the Zoo modernization. This request was granted. The Doctor then spent many hours in consultation with the architects and engineers working out the details. The overall plan thus produced has been recently substantially approved by the City Administration and within the next few years Dr. Cadwalader's entire dream will be realized." (Charles I. Thompson).

Bill's attainments in all of his different fields of interest were always of the highest order, but the outstanding quality that was instinctively realized no less by those who were merely honored by his acquaintance, than by the fortunate many who could claim him as a friend, was the ever-present, innate, sterling quality that characterized Williams Biddle Cadwalader—the man! For indeed, we shall not look upon his like again!

In closing, we may appropriately give voice to the old Attic Greek salutation for triumph or victory: Euge—Well Done, Bill!

Memoir of John Stewart Rodman (1883–1958)*

By CALVIN M. SMYTH, M.D.

JOHN STEWART RODMAN was born on July 21st, 1883 in Abilene, Texas, and died at his home in Radnor, Pa. on April 26, 1958. His parents, William L. Rodman and Bettie Stewart Rodman, at the time of his birth were stationed there during a period of duty with the Army. Much of his early life was spent in Louisville, Kentucky where his father was Professor of Surgery in one of the five schools of medicine which flourished there at that time. The family moved to Philadelphia when the elder Dr. Rodman was appointed to the Chair of Surgery at the old Medico-Chirurgical College. After graduating from the William Penn Charter School, he entered the college department of the University of Pennsylvania and, after completing his pre-medical studies there, received his M.D. degree at Medico-Chi in 1906. An internship at the Pennsylvania Hospital was followed by a fellowship at the Mayo Clinic.

He married the former Eunice B. Hinman. They had two children, William L. Rodman and Mrs. Eunice Rodman Packer, who with his widow survive him.

Dr. Rodman had an unusual career in surgery. His father was the actual originator of the idea that there should be one examining body whose certificate would admit successful candidates to practise anywhere in the United States. He became firmly convinced of this when he was required to take the examinations of the Pennsylvania State Board when he came to Philadelphia as a Professor. Through his efforts the National Board of Medical Examiners was constituted. When the elder Rodman died in 1915 his vision had not become much of a reality. Stewart Rodman, as an act of unusual filial devotion, dedicated himself to the accomplishment of his father's dream. This was a long and up hill fight. For a young surgeon in the course of attempting to develop a surgical practise in a place like Philadelphia this was a very real sacrifice. He became the National Board's first Secretary and Treasurer, a position which he filled for many years. This took a tremendous amount of time and called for repeated and sometimes prolonged absences from the city; unquestionably interfering with any clinical career in surgery. He did, however, manage to place the National Board in the secure position which it presently occupies; although its original objectives have been only partially attained.

In 1924 he was appointed to the Professorship of Surgery in the Woman's Medical College of Pennsylvania which position he filled with distinction until he reached academic retirement age. He was an excellent teacher, meticulous in the preparation and delivery of his lectures.

Dr. Rodman's outstanding contribution to American Surgery was his setting up and conducting the administration of the American Board of Surgery. He became the first Secretary of the Board, and continued in that position from the founding of the Board in 1937 until his retirement in 1953. Only those who were closely associated with him in the organizational period and early years of the Board can appreciate the work that this entailed. Extensive travel for the holding of the final part of the examination was required. Much work went into the setting up of the examination. In later years this amounted to as many as eleven trips a year, covering the country from coast to coast, and often requiring absences of a week at a time. The conduct of the office of the Board was something which required infinite patience, and an appalling

* Read before the College of Physicians of Philadelphia, 5 November 1958. Prepared and published at the request of the Council of the College of Physicians of Philadelphia.

amount of correspondence. The position inevitably made for hard feeling on the part of candidates for examination who could not understand why their training did not meet the Board's requirements. The candidate who failed the examinations not infrequently made a personal issue out of his failure. In spite of the many headaches connected with the position, I know that Dr. Rodman considered this the most rewarding experience of his entire professional life. The profession recognized his accomplishments by election to the American Surgical Association as a Fellow and later as Vice President. He served as Secretary and President of the Philadelphia Academy of Surgery. He was a Fellow of the American College of Surgeons and of the Philadelphia College of Physicians. In addition to his Professorship at the Woman's Medical College, he was at one time Surgeon-in-Chief to the Bryn Mawr Hospital, and had been on the Staff of the Presbyterian and the Philadelphia General Hospitals. He was a member of the Philadelphia Club, the St. Anthony Club, and the Fraternity of Delta Psi. His principle contributions to the literature were a number of papers on cancer and other surgical conditions of the breast.

Throughout the last twenty years of his life, Dr. Rodman was plagued by ill health. For many years he periodically suffered intensely from a duodenal ulcer for which he eventually underwent subtotal resection with a most happy result. His freedom from gastric distress, however, was shortly followed by prostatic difficulties and then a coronary occlusion. He died as a result of another coronary attack, his third. Throughout these years his life was made richer by the loyal devotion of his wife, and their marriage was one of the happiest possible.

Stewart Rodman's death leaves an empty place in the surgical scene in America and in the hearts of his friends. He was a great gentleman, a loyal friend, and a good companion. May he rest in peace.

College Notes

DR. CRAMPTON RECEIVES FRANKLIN INSTITUTE MEDAL

Dr. George S. Crampton, Professor Emeritus of Ophthalmology at the University of Pennsylvania School of Medicine, recently received an Edward Longstreth Medal from the Franklin Institute for his development of the borescope, a specialized periscope for the internal inspection of turbine shafts and various closed vessels.

Dr. Crampton was awarded the medal Wednesday, October 15, 1958, at formal ceremonies in the Institute's Franklin Memorial Hall.

The medal citation to Dr. Crampton reads: "For his development of the Borescope, a specialized periscope for the internal inspection of turbine shafts, rotors, guns and various closed vessels, by which flaws may be detected thus providing protection of vital importance from the physical and economic hazards of operational failure." Dr. Crampton has been awarded two patents, one for the borescope and the other for Trial-Lens Mountings for oculists and opticians.

Dr. George S. Crampton, Professor Emeritus of Ophthalmology at the University of Pennsylvania, receives an Edward Longstreth Medal from the President of The Franklin Institute, Wynn Laurence LePage, for his development of the borescope. Dr. Crampton and sixteen other men received their awards at formal ceremonies on October 15, 1958, at The Franklin Institute, Philadelphia.

The Longstreth Medal, founded in 1890 by Edward Longstreth, is awarded by the Institute, Philadelphia's 134 year-old scientific-educational organization for inventions of high order and for particularly meritorious improve-

ments and developments in machines and mechanical processes.

Dr. Crampton, who has developed many specialized types of borescopes, was born on March 10, 1874, in Rock Island, Illinois. He was graduated from the Medical School of the University of Pennsylvania in 1898. He traveled and studied abroad for several more years. On his return, before entering the Pennsylvania Hospital as resident physician (1904–06), Dr. Crampton did research in its Ayer Clinical Laboratory for eighteen months. Later he was a member of the staff of that hospital as well as Wills Eye Hospital. During World War I, he served as Director of the four field hospitals of the 28th Army Division. He joined the staff of the University of Pennsylvania Graduate School of Medicine in 1919.

In 1920, in response to a request by Westinghouse Electric Company which knew of his interest in electrical engineering, Dr. Crampton developed the first borescope in the attic of his Philadelphia home. That same year, he established the Lenox Instrument Company to produce the instrument used in various forms to detect flaws in guns, aircraft propellers, gas turbine engines, etc. Dr. Crampton also designed and made the first borescope for the Manhattan (A-Bomb) project.

Dr. Crampton holds membership in the Optical Society of America, American Ophthalmological Society, College of Physicians of Philadelphia, and the Illuminating Engineering Society, (national president, 1920–21). Dr. Crampton was awarded the Gold Medal of the I.E.S. at its Fiftieth Technical Conference in 1956.

JOURNAL ISSUES WANTED BY COLLEGE LIBRARY

The journals listed below are lacking from the files of the Library of the College of Physicians of Philadelphia. If any readers of the *Transactions & Studies* have copies which they are willing to donate to the Library they will be most gratefully received.

Algérie Médicale.	v. 58, no. 11, 1954.
	v. 62, no. 1, 1958.
Boletin de la Sociedad de Cirugía del Uruguay.	v. 27, nos. 1, 4, 5, 1956.
British Journal of Radiology.	v. 25, 1952.
Bulletins et Mémoires de la Société Française d'Ophtalmologie.	v. 68, 1955.
Clinical Chemistry	v. 2, no. 4, 1956.
Fiziologicheskii Zhurnal USSR.	v. 39, no. 1, 1953.
Folha Medica.	v. 35, nos. 7, 11–14, 1954.
Gaceta Medica de Caracas.	v. 60, nos. 3–4, 1953.
	v. 61, nos. 11–12, 1954.
Gazzetta Sanitaria.	v. 28, no. 1, 1957.
Gynécologie Pratique.	v. 4, no. 2, 1953.
Journal of the Canadian Association of Radiologists.	v. 1, no. 1, 1950.
Journal of the Formosan Medical Association.	v. 56, nos. 5–6, 11–12, 1957.
Medicina. Buenos Aires.	v. 16, no. 4, 1956.
Medicina, Cirurgia, Farmacia.	1955, nos. 226–227.
Resident Physician.	v. 2, no. 5, 1956.
Revista de la Facultad de Medicina. Bogota.	v. 20, nos. 10–12, 1952.
Science Tools.	v. 1, no. 1, 1954.
State of Mind.	v. 1, no. 4, 1957.
Vestnik Khirurgii.	v. 74, nos. 1–4 1954.

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*President's Address**

By JONATHAN E. RHOADS, M.D., D.SC. (MED.)

IN THIS annual address, I will attempt to set forth the state of the College as objectively as possible, both in absolute and relative terms. The factor of time leads me to restrict this review to three phases of the College's operations: finance, library and membership.

The assets of the College will be presented in detail in the Annual Report of the Treasurer. Our total assets exclusive of our book stocks are estimated at $2,718,337.66. The land, buildings and fixtures are appraised for insurance purposes to be worth $1,588,747.93. Some estimates have been higher. The endowment and current funds of the College had a book value of $1,117,196.15 on November 11, 1958, and a market value of $1,387,370.11 on the same date. The book stocks cannot easily be appraised, but their replacement cost would probably exceed all of the other assets of the College combined. Thus, our predecessors have passed on to us a trust worth between $5,000,000. and $10,000,000.

Additions to our capital in the past year lie partly in the library's acquisitions, where we have invested $25,125.52 in books and journals and the binding thereof, and have added gifts and exchanges of 1,486 volumes.

Through the efforts of our former President, Dr. T. Grier Miller, the College has received over $15,500 from 27 donors for new Chairs and for the support of the library. We also record receipt of notice of two bequests. Under the will of Mrs. Celia Bolles Nicholson, widow of our deceased Fellow, Dr. William R. Nicholson, the College receives the income of one-quarter of his residuary estate, and later after the death of a life tenant, an additional one-quarter of this income. It is estimated that this will be the equivalent of a bequest of approximately $10,000. Under the will of our late Fellow, Dr. William Zentmayer, the Library will receive $15,000. and possibly one-ninth of the residuary estate will come to the College at a later date.

The work of the Curator is currently being supported by a grant from the American Philosophical Society of $13,000., $6,000. of which was received August 1, 1958—the balance to be sent February 1, 1959—and this is being utilized to catalogue our rare book collections.

The Librarian was successful in obtaining a number of worthwhile grants with the result that the Library had nearly $7,000. on hand at the end of the fiscal year toward its current budget. Smaller donations provided for replacement of the Officers' gowns. A modest deficit in the general operating budget of $1,345. was shown at the end of the fiscal year. The operation of the Erlanger Garage not only paid the income formerly received on the

* College of Physicians of Philadelphia, 7 January 1959.

funds used for its purchase, but produced $1,549.78 toward its amortization. A new heating plant may have to be procured, and if so would constitute an additional capital investment.

A precise estimate of what additions to the total assets of the College amount to cannot be made due to uncertainties in the value of bequests and the value of books, but it would appear to be of the order of $75,000. to $100, 000.

The annual operating expenses for 1957–1958 were $157,081.35. The operating budget adopted for 1958–1959 is $178,075.12. Income estimates lead us to believe that the budget will be in balance. Thus, while the past year has not brought a major increase in the College's assets, there has been steady growth and the financial position of the College appears to be sound with physical assets in excellent condition.

Our Library passed a milestone about December 1, 1958, and has over 200,000 accessioned items. This compares with 1,000,000 for the National Library of Medicine, with 302,000 for the Library of the New York Academy of Medicine, and with 231,607 for the Library of the Boston Medical Library as of 1958. We subscribe to over 2,000 serials. Our most valuable collections have been transferred to a locked area on the upper floors of the new stacks.

The Medical Documentation Service has expanded its activities and is operating well in the black and making important industrial friends for us, as well as serving our own Fellows.

Finally, I wish to report on changes in our membership. During the past year, we have lost through death 24 Fellows, through resignation 8 Fellows, and through non-payment of dues 4 Fellows. Those received into Fellowship number 64, so that the total Fellowship of the College numbers 1,160, composed of 1,016 Active Fellows, 134 Non-resident Fellows, 8 Honorary Fellows, and 2 Honorary Associates. It is believed that the total number of physicians in greater Philadelphia is approximately 5,500, and that the proportion having Active Fellowship in the College is probably lower than at various times in the past.

Among new, Active Fellows we list 2 Deans, 7 Professors, 17 Associate Professors, 18 Assistant Professors, and 8 others with teaching appointments. Most of the new Fellows have contributed to the medical literature, and all of them have been recognized as respected members of the profession in their several communities. It is my belief that the new members continue in the time-honored tradition of the College, and that the increased number reflects a larger number of highly qualified physicians in the area on the one hand, and a more careful search for highly suitable persons, many of whom have been overlooked in the past, on the other hand.

Your President is deeply indebted to his fellow officers, Dr. Thomas Durant, Vice-President; Dr. Thomas Machella, Secretary; and Dr. Jacob Vastine, Treasurer; to the Censors, to the past Presidents who have remained unusually active, to the members of Council and the 57 Fellows who have served as the Committees or as Officers of Sections of the College. The Staff of the College has served it very well under the leadership of Mr. Elliott Morse, Librarian; Dr. W. B. McDaniel, Curator of rare books; Miss Ella Wade, Curator of the Mutter Museum; Mr. George Allen, Superintendent of the buildings; and Miss Olga Lang, Clerk of the College. They and their several assistants have operated the College for us.

As the College faces the future, should its purposes either change or undergo a shift in emphasis? If we accept its basic purposes as a joint effort of this medical community to improve professionally not only its membership, but the entire medical profession for the greater good of the society we serve, I believe that we would want to reaffirm this central purpose. However, when we review the specific and detailed objectives of the various programs instituted to further our basic purpose, there may be more room for revision.

Our Museum is constantly adding to the College collections, and these items become progressively more valuable with the passage of time. It would seem to me that this activity was being very capably conducted by the Curator and by the committee of fellows in charge, and that it was probably receiving about the right amount of emphasis at the present time.

With respect to the Library, we are almost certainly facing progressively higher costs. Salary levels of librarians generally are low, and we are somewhat low among competing libaries. As far as one can see, the only direction in which these costs can go is up, and the questions are: how far and how fast? Costs of binding and costs of journals and books are constantly mounting, and we are not adding as aggressively as we should be to our historical collections. Broadly speaking, the costs of the Library are two parts for staff and one part for books. Despite the popular trend away from endowments, I believe it is extremely important for the College to increase the endowment for its Library. If we fail to do this, I believe that within a generation we will yield to pressures which we do not wish to yield to, in order to gain the necessary support for this activity. I am not overly optimistic about raising major sums of money from the public nor from foundations for this purpose. I think our great hope lies in the possibility that those of our Fellows who accumulate some of this world's substance will remember the College in their wills and thus leave the College a sum sufficient to yield an annual income of $100., $500., $1,000. or more. During the past year, as I mentioned, two such bequests have come to the College along with certain smaller amounts.

The possibility of governmental support has been explored with the Surgeon General of the Public Health Service, and apparently there is no opportunity for financial support of research libraries at present. It would seem that such support would be logical and I hope that an opportunity may arise to raise this question with members of Congress.

With the promulgation of the Medical Documentation Service, the College is serving industrial users much more than formerly, and the Library's revenue from this source has increased sharply. The question of whether the borrowing privilege should be extended to members of other medical societies and specifically to the County Medical Society is a significant one. My own belief is that if the County Society with its membership of 3,200 physicians wished to acquire this privilege and was willing to pay what seemed to be a realistic sum for the privilege, it would be wise for the College and in accordance with its basic purposes for this privilege to be extended. This would not include the right to sign cards for others, which would remain the special prerogative of Fellows of the College. Fellows of the College would continue to enjoy preferential treatment in the purchase of additions to the Library in which they are interested.

The question naturally arises as to what is a realistic sum. My own view is that this should be of the order of $7.50 per member of the County Society on a 100 per cent enrollment basis. It should be recognized that this will cost the College something in terms of personnel to service the new demand; it will cost it something for re-binding and replacement of worn volumes; it will save the County Society an estimated $5,000. or $6,000. a year plus overhead for its present library; and it will provide for members of the County Society a very much finer library service. In any such negotiation, I think there should be an escalator clause, so that as the costs of our Library's operation rise, the membership of the County would share more or less proportionately with the Fellows of the College the increased cost. It is, of course, uncertain whether the County Society would have any interest in such an arrangement which might necessitate an increase in the annual dues.

Turning to our other great educational venture, our lectureships, I believe we should recognize that these lectureships when originally set up provided an attractive payment to the lecturers. In general, these payments range from $100. to $200. plus expenses. We have

continued to have a very gratifying response from persons invited to lecture at the College, largely I believe because of the eminence of those who have given these lectures over past years. The customary fee for lectures by distinguished individuals in academic circles has now increased, so that $200. or $250. is more or less minimal, and the more financially attractive lectureships pay from $300. to $500. The problem is whether we can raise funds to augment the endowments of these lectureships without changing the names. It may prove easier to raise funds for new lectureships and simply to give the older named lectureships less frequently, allowing the income to accumulate up to two or three years if necessary, in order to provide an attractive stipend.

A field in which the College might play a considerably larger role than it has lies in inviting distinguished foreign medical scientists to this country. Because of currency regulations and the economic structure under which the medical profession labors—particularly in the British Isles, it is difficult for our colleagues from England and Scotland to travel without fairly remunerative lectureships. It would be a splendid thing if the College could command the resources to bring 2 to 6 distinguished foreign physicians or medical scientists to the Philadelphia area each year, arranging for them to give such of our named lectureships—as might seem appropriate—and making them all available for visits to the five medical teaching institutions in this city and some of the independent research institutes in the area. This would essentially be a new program and rather an expensive one, but I think it could have great meaning for the enrichment of the programs of our teaching institutions, for increasing the contacts between Fellows of the College and their colleagues abroad, and that it would be a good thing for Philadelphia medicine generally.

Dr. Lewis Scheffey has had a special interest in the program of International Visitors, which has brought a number of worthwhile guests to the College on an informal basis. These people carry away a strong impression of one of the city's important cultural institutions and not infrequently these visits lead to stimulating contacts with individual Fellows.

Access to the College by automobile is becoming better and better with the completion of the Schuylkill Expressway. Parking facilities remain excellent because of the large lot across Twenty-second Street and the parking garage which backs up to the College and which we own. If, as we fear, the lot across Twenty-second Street should be used for building purposes, we can rely on parking in the garage behind us. The present lease on this garage expires in a little over two more years, and it would be my hope that the lease could be renewed with a provision which would insure priorities for Fellows of the College in the event of a parking stringency. It is quite possible that some money will have to be spent at such time as the lot across Twenty-second Street is taken over for other uses in order to make better use of the upper floors of the parking garage. If Fellows' cars have to be parked above the first floor, the present elevator service will be rather slow. It is my understanding that the garage formerly had a ramp to the second floor, and it may well be necessary to have this re-installed. It is to be hoped that the garage will produce enough revenue to provide for these improvements, but it is possible that some additional investment may be required.

Perhaps the most encouraging thing of all during the past year has been the increased use factor of the College and its programs. A downward trend in the number of books taken out which has continued for several years past, has been reversed and the Library shows an increased usage in all categories. While not all of the meetings of the College have been well attended, the past fall has seen one of the largest audiences ever to occupy Mitchell Hall. Your officers believe these signs are encouraging and we seek your suggestions as well as your support for the continued operation of our College.

REPORT OF THE TREASURER FOR THE YEAR ENDED NOVEMBER 10, 1958

Assets

General fund		
Cash in bank and on hand		$11,402.13
Maintenance and reserve fund		
Cash in bank	$1,213.47	
Investments, at book value	10,287.76	11,501.23
Miscellaneous contributions fund		
Cash in bank		16,760.99
Library historical collections fund		
Cash in bank (restricted)		6,000.00
Trust funds		
Uninvested principal cash	9,292.47	
Uninvested income cash (restricted)	28,265.95	
Loan receivable—garage equipment	935.24	
Mortgage	45,000.00	
Real estate	154,421.10	
Bonds	294,825.90	
Preferred stocks	257,851.63	
Common stocks	354,869.81	1,145,462.10
Plant fund		
Land—15–25 South Twenty-Second Street	95,000.00	
Building	1,420,747.93	
Furniture and fixtures	1.00	
Deposits on perpetual fire insurance policies	11,462.28	1,527,211.21
Total		$2,718,337.66

Funds

General fund			
Library general account		$6,952.96	
Life membership account—Uninvested portion		674.00	
Reserve for contingencies—Uninvested portion		190.02	
Unexpended operating fund balances			
Current operations	$2,301.94		
President's room	525.00		
Friends of library	20.00		
Books	387.35		
Rare books	152.96		
Campaign expense	165.95		
Portrait	126.47		
Entertainment	107.66		
Harvey's "De Motu Cordis" Facsimile 1628 Edition—overexpended	(202.18)	3,585.15	$11,402.13
Maintenance and reserve fund			
Principal net worth			11,501.23
Miscellaneous contributions fund			
Principal net worth			16,760.99
Library historical collections fund			
Principal net worth			6,000.00

Trust funds			
Principal net worth, at book value			
Discretionary common trust fund	1,033,157.15		
Legal common trust fund	24,097.94		
Trust funds not pooled	14,941.06		
Fund for purchase of units in discretionary common trust fund	45,000.00	1,117,196.15	
Income net worth			
Discretionary common trust fund	26,064.82		
Legal common trust fund	693.54		
Trust funds not pooled	1,507.59	28,265.95	1,145,462.10
Plant fund			
Principal net worth			1,527,211.21
Total			$2,718,337.66

General Fund
Statement of Receipts and Disbursements
November 11, 1957 to November 10, 1958

Receipts		
Income on investments, net of agent's fees		
Trust funds	$60,069.56	
Income funds, principal of which is not administered by the College of Physicians of Philadelphia	1,260.64	
Mortgage	2,088.28	$63,418.48
Contributions		
Fellows dues	55,793.00	
Life membership	1,719.00	
Entrance contributions	2,550.00	
Use of halls and projector	2,844.00	
Other	100.00	63,006.00
Library		45,601.46
Miscellaneous		2,998.22
Total receipts		175,024.16
Disbursements		
Operating expenses		
Hall committee	29,465.57	
Office of the Secretary	6,371.48	
Office of the Treasurer	8,239.87	
Pensions	2,523.14	
Campaign expense	10.00	
Publication committee	5,397.74	
Books	1,715.70	
Entertainment committee	798.14	
College collections committee	3,220.35	57,741.99
Library		
Salaries and payroll taxes	56,649.12	
Books, journals, supplies, etc.	39,690.41	96,339.53

Restricted trust funds		
Discretionary common trust fund	5,083.80	
Legal common trust fund	330.26	
Trust funds not pooled	1,358.55	6,772.61
Total disbursements		160,854.13
Excess of receipts over disbursements		$14,170.03

Notes: Receipts for the year ended November 10, 1958 of special purpose funds from contributions and security investments not included in the above schedule were as follows:

Fund	Amount
Maintenance and reserve fund	$396.49
Miscellaneous contributions fund	16,760.99
General endowment fund	1,985.00
Library historical collections fund	6,000.00
Book of remembrance	370.00
College extension fund	320.00

ANNUAL SUMMARY STATEMENT

Endowment and Extensions Funds of the College of Physicians of Philadelphia

General Endowment Fund
Statement of Receipts and Disbursements
November 12, 1957 to November 11, 1958

Balance, November 12, 1957		$347,521.43
Receipts		
Entrance contributions	$2,550.00	
Gifts from Fellows (Voluntary contributions)	2,015.00	
Gifts and bequests (Note 1)	1,523.68	
Gifts from Book of Remembrance	370.00	
Amortization of mortgage—13th and Locust Streets	2,250.00	
Amortization of loan—garage equipment	324.00	
	9,032.68	
Add: Net addition, current year, from the unit adjustment account	2.72	9,035.40
Balance, November 11, 1958		$356,556.83

Notes: (1) Gifts and bequests were received as follows:

Estate of Joseph Walsh	$1.04
Estate of Horace Phillips	522.64
Estate of William Penn Vail	1,000.00
Total	$1,523.68

The General Endowment Fund also received a gift of five shares, common stock of the Chase Manhattan Bank from Samuel M. Freeman.

Restricted Endowment Funds
Statement of Receipts and Disbursements
November 12, 1957 to November 11, 1958

Discretionary common trust fund		
Balance, November 12, 1957		$534,013.50
Receipts		
Paul Judd Sartain Library Fund	$4,970.13	
Pension Reserve Fund	547.44	
Life Membership Fund	1,100.40	
	6,617.97	

Less: Amounts insufficient to purchase one unit credited to the unit adjustment account	24.57	6,593.40
Balance, November 11, 1958		540,606.90
Legal common trust fund		
Balance, November 11, 1958 (no change during the year)		23,500.00
Trust funds not pooled		
Balance, November 12, 1957		14,920.93
Receipts		
Sale of stock rights		20.13
Balance, November 11, 1958		14,941.06
Total Restricted Endowment Funds, November 11, 1958		$579,047.96

College Extension Fund
Statement of Receipts and Disbursements
November 12, 1957 to November 11, 1958

Balance, November 12, 1958		$1,212.48
Receipts		
Gifts from Fellows (Voluntary contributions)	$320.00	
Income earned on invested principal	59.88	
	379.88	
Less: Amount insufficient to purchase one unit credited to the unit adjustment account	9.22	370.66
Balance, November 11, 1958		$1,583.14
Total Funds at book value, November 11, 1958		$937,187.93

JACOB H. VASTINE, 2nd, M.D.
Treasurer

Annual Report of the Library*

ADMINISTRATIVE

Serials Traffic Pattern

THE MOST volatile item of the medical library's stock in trade is the serial publication. A share of the budget, second only to salary, is devoted to the acquisition, binding, housing, and circulation of these prolific evidences of medical research activity. Psychological studies have been made of the research habits of scientists and a new technical vocabulary has been developed to redefine the process of extracting specific bits of information from this ever-flowing stream of facts and conjecture. As intimately as we live with these paper and ink fruits of the medical press we know relatively little about the factors which cause their relative popularity.

Nose counting projects such as the one being reported here and the one described in the 1952/53 report are helpful as indications of the most efficient physical location of material but they do not provide sufficient information for precise forecasts of future traffic patterns. The 1952/53 study did provide sufficient evidence, however, to strengthen the decision to transfer all journals published before 1940 to the newly constructed stack addition which is more remote from the reading rooms, leaving space for growth in the original stack unit which is closer.

Also derived from this study was a list of the 50 most requested titles. This list was based upon the total number of requests for each title regardless of the date of the issue requested. This popularity poll was then rather illogically used as a means of determining which journals should have their most recent bound volumes shelved in the Reading Room in order to locate the most used material in the most strategic position. Overlooked was the fact that long-established journals earned statistical advantages through the continuing demand for back volumes.

Objectives. In recognition of the possibly fallacious application of the earlier study and the fact that relative popularity in 1952/53 may differ from that of 1957/58, another study was undertaken of the circulations during the current report year. It is hoped that this study provides a valid basis for the determination of the titles to be shelved in the Reading Room and perhaps also for the selection of that one third of the library's currently received titles which can be housed in the Periodicals Room. The statistical tabulations have also been examined in the expectation that they may show how long the period of maximum demand lasts. These factors are also pertinent to the choice of those titles for which reserve copies will be kept and to the decision of the length of time such reserve files should be maintained. It has also become a matter of curiosity to discover what relationship there may be between the relative popularity of journals based on the circulation of quite recent volumes and their popularity based on total circulation regardless of date.

Heavy Demand Period. In order to select the years upon which the popularity poll would be based, an effort was made to determine the period of heaviest demand. The circulation of 1950 volumes was tabulated by year while the remainder of the twentieth century circulations was tabulated by decade. A separate scoring was kept of the pre-1900 circulations. This part of the study was limited to the circulations of 167 different titles whose total circulation was 17,375. This represents about one third of the year's total journal circulation of 53,831. The remaining two thirds is made up of scattered calls made for a great many titles no one of which was called for any considerable number of times. The following traffic pattern is revealed by this statistical maneuver:

* 1 November 1957–31 October 1958.

1958	620
1957	2492
1956	1894
1955	1578
1954	1185
1953	971
1952	779
1951	649
1950	696
	10,864

1950's	10,864
1940's	3,427
1930's	1,818
1920's	782
1910's	234
1900's	126
pre-1900's	124

The only statistically significant drop in these figures occurs after the peak circulation is reached in the period between one and two years after publication. The decline between 1957 and 1956 is about one third. The average drop during the rest of 1950's is about twenty percent with a twenty-five percent drop between 1955 and 1954. The year 1950 bucks the trend and shows a slight increase. This peculiarity was fairly generally observed and did not seem to result from one reader's interest or from the circulation of any small number of titles.

As would be true of any current year's figures, the 1958 count is low because the 1958 issues were appearing during the report year. In many cases these journals were used directly from the shelves in the Periodicals Room with no record being kept of their use.

These figures taken together with the circulation figures for the 100 most called for journals may suggest an answer to the question whether the available space in the Reading Room is best used for longer runs of fewer titles or shorter runs of more titles.

"*Your Hit Parade*". The following list gives the titles of the first 100 journals in the order of their popularity as shown by the number of calls made during the report year for volumes in the 1955 to date period.

J.A.M.A.	181
Ann. New York Acad. Sc.	149
Lancet	141
Brit. M. J.	131
New England J. Med.	131
Proc. Soc. Exper. Biol. & Med.	129
Am. J. Med.	105
Nature	97
New York J. Med.	83
Ann. Int. Med.	83
A.M.A. Arch. Surg.	73
Canad. M. A. J.	70
J. Exper. Med.	69
A.M.A. Arch. Int. Med.	67
J. Clin. Invest.	67
Am. J. Clin. Path.	62
Science	62
Med. Clin. N. Am.	61
Pediatrics	61
Proc. Roy. Soc. Med.	61
Am. J. Clin. Nutr.	59
M. J. Australia	59
Am. J. Physiol.	58
J. Chron. Dis.	58
Am. J. Obst. & Gynec.	57
Am. J. Surg.	57
Am. J. Roentgenol.	56
Am. J. Ophth.	55
J. Lab. & Clin. Med.	55
Am. J. M. Sc.	54
Circulation	54
J. Biol. Chem.	54
J. Clin. Endocrinol.	54
J. Physiol.	54
Obst. & Gynec.	54
South. M. J.	53
J. Am. Pharm. A. Scient. Ed.	50
A.M.A. Arch. Path.	48
J. Am. Dietet. A.	47
J. Urol.	47
California Med.	46
J. Biophys. & Biochem. Cyt.	46
Surg. Clin. N. Am.	46
J. Nat. Cancer Inst.	45
Surgery	45
Ann. Surg.	44
A.M.A. Am. J. Dis. Child.	43
Am. J. Pub. Health	43
Arch. Biochem.	42
Canad. J. Biochem. & Physiol.	42
Cancer Res.	42
J. Invest. Dermat.	42
Nutrit. Rev.	42
Anat. Rec.	41
Biochem. J.	41
J. Pediat.	41
Surg. Forum	41
A.M.A. Arch. Neurol. & Psychiat.	40
Am. Heart J.	40
Biochim. & Biophys. Acta	40

Postgrad. Med.	40
Pub. Health Rep.	39
Am. J. Psychiat.	38
Gastroenterology	38
Indian J. Med. Res.	38
J. Clin. Path.	37
Med. Times	36
Minn. Med.	36
Am. J. Path.	35
Arch. internat. pharmacodyn.	35
Arch. klin. u. exp. Derm.	35
C. R. Acad. Sci.	35
Presse méd.	35
Antibiotics ann.	34
Cancer	34
Endocrinology	34
J. Int. Coll. Surg.	34
J. Mt. Sinai Hosp.	34
Klin. Wchnschr.	34
Acta endocrinol.	33
Acta med. scandinav.	33
Blood	33
J. Comp. Neur.	33
Northwest Med.	33
Practitioner	33
A.M.A. Arch. Derm. & Syph.	32
Ann. Rev. Physiol.	32
Laryngoscope	32
Schweiz. med. Woch.	32
West. J. Surg.	32
Experientia	31
J. Am. Geriatrics Soc.	31
J. Lancet	31
Am. Surgeon	30
Ohio M. J.	30
South African M. J.	30
A.M.A. Arch. Ophth.	29
Am. Rev. Tuberc.	29
Arch. Dis. Childhood	29
J. Immunol.	29

Breadth or Depth. Although many variables may produce different conclusions in other library situations, these figures suggest a principle which some statistically minded observer may be able do reduce to a neat formula. The problem to which such a formula might be applied is whether it is better to use available Reading Room shelving for longer files of a relatively few titles or shorter files of a greater number of titles.

The College library has been keeping about seventy titles in the Reading Room and was recently obliged to shift the starting date of these files from 1950 to 1955 to keep the collection within bounds. The present study would seem to indicate that if the number of titles shelved in the Reading Room from 1955 to date were increased from seventy to one hundred there would be a saving of about 900 trips to the Stack in a year. At the present time there would be about seven volumes for each title which would mean that about 210 volumes would be moved into the Reading Room from the Stacks.

The 1954 issues of the first seventy titles on the above list circulated 306 times during the year under study. It may be presumed therefore that changing the starting date of these files from three years back to four years back would save this number of trips to the Stacks. One additional year of the seventy titles presently shelved in the Reading Room would add about 175 volumes to the Reading Room.

For this particular library this would mean that four trips to the Stacks would be saved for every volume of the fourth year back shelved in the Reading Room whereas 4.3 trips might be saved by each volume of the next 30 most popular journals which could be shelved in the Reading Room. Because the Reading Room can accommodate about 1125 volumes, this fairly narrow margin of difference could be a very appreciable factor suggesting an advantage can be gained by expanding the list of titles shelved in the Reading Room.

Old Timers' Influence. The results of the popularity poll published in this report were based, as has been noted, on the circulation of volumes published in 1955 and later. There would have been somewhat different results if the list had been compiled on the basis of total circulation regardless of the date of publication. Space considerations argue against the printing of the first one hundred titles on the basis of such a scoring system. However, the following listing of the first ten titles shows the effect of the existence of long back files upon such a study:

J.A.M.A.
Proc. Soc. Exper. Biol. & Med.
Lancet
J. Biol. Chem.

Brit. M. J.
Am. J. Physiol.
J. Physiol.
New England J. Med.
Am. J. Obst. & Gynec.
J. Exper. Med.

It may be noted that five of these titles have been displaced from the first ten in the list of 1955 to date circulations. Four of these five began to be published in the nineteenth century. There are only two journals in the second listing which began after the turn of the century: *Proc. Soc. Exper. Biol. & Med.* (1903), and *J. Biol. Chem.* (1905). The durability of the veterans is attested to by the fact that only four twentieth century entrants are in the first listing. *Proc. Soc. Exper. Biol. & Med.* (1903) appears on both lists. The *New York State Journal of Medicine* whose 1901 starting date barely qualifies it to be considered as a twentieth century journal is now in the first ten. The other brash newcomers to the charmed circle are *American Journal of Medicine* (1946) and *Annals of Internal Medicine* (1927).

Because of the fact that this library receives only the medical issues of the *Annals of the New York Academy of Sciences*, which are then bound separately, it is true that calls for one year's file of this title result in a larger number of circulations than in the case of other titles.

Among the other variable factors which have an influence upon such a study, mention might be made of the obvious fact that journals appearing weekly enjoy an advantage over journals which appear less frequently because of the greater number of articles which will appear in the literature. Five of the journals in the first ten titles are weeklies.

This study does not pretend to explore all of the factors which contribute to the relative popularity of medical journals. The fact that a journal is distributed to all of the members of a society, questions of editorial policy, and the degree of specialization of the articles would all provide interesting side excursions.

The short range objectives of this study will have been achieved if it has provided valid bases for the physical arrangement of the journal files and for the selection of titles for which reserve files should be maintained. A bench mark may have been established for future comparisons and questions left unanswered by this review may attract another investigator's attention.

The College Library in 1957/58

Deepening the Roots. Several developments during the year covered by this report may prove to have far-reaching effects on the future progress of this institution. Growth is so frequently superimposed upon a rigidly confined structure that it is especially rewarding to witness events which may serve to strengthen the foundations themselves.

Medical School Support. The need for broadening the basis of financial support for the independent learned society library has been a familiar Leitmotiv of these reports at least as far back as 1953. It is understandable therefore, that a considerable amount of enthusiasm greeted the generous financial grants which were made voluntarily to the College library during the year by the local medical schools. Gifts were received from Hahnemann Medical College, Woman's Medical College, Jefferson Medical College, Temple University School of Medicine, and the Graduate School of Medicine of the University of Pennsylvania. This local recognition of the regional value of the College library and the willingness to share in the efforts to increase its usefulness are most gratifying. Certainly medical research is strengthened by the cooperative support of a scientific library devoted to serving the entire medical community.

Job Descriptions and Salary Scale. A long stride forward was taken toward the stabilization of library personnel problems by the Library Committee's careful study and approval of the principle of established job descriptions and salary scales. Contingent upon satisfactory job performance and budgetary considerations, it is recognized that the acceptance of the principle of regular salary increments within established ranges provides a valid basis for long-

range fiscal planning and encourages staff continuity.

New Charging Desk. The long-cherished dream of a new circulation desk large enough to accommodate more than one Reading Room attendant and more than one telephone was finally realized this year. The two-man operation of this library nerve center during rush periods has contributed to the improvement of service to readers in the room and to those who call by telephone. The removal of a constant cause of annoyance has materially aided staff morale.

New Circulation Forms. A new multiple copy circulation slip with interleaved carbon paper was put into use during the year. This new form eliminates the necessity for painstaking transcription of information by hand. The double record needed to show who has taken a particular book out as well as what books are charged to a particular borrower is now provided when the original request slip is written by the borrower. This form has been adopted by several local libraries to simplify their procedures for borrowing volumes from the College library. By using a three-part form, they have a record of the titles requested while they supply the College library with slips which can be filed directly into the circulation desk records.

Photoreproduction Prices. The charges made by the library for the photoreproduction of library materials were revised during the year to more nearly reflect the costs of providing this service. The elimination of this item of deficit budgeting has eased tight spots in other categories.

Medical Documentation Service Directorship. The appointment of a full-time salaried Director for the Medical Documentation Service is recorded in another section of this report. However, the library administration wishes to record its special appreciation to the clients of the service who encouraged the College to establish a fee schedule that made this appointment possible and also to make a matter of record its pleasure over the appointment of a man of Mr. Robert Blanken's abilities to the Directorship.

American Philosophical Society Grant. This chronicle of dreams come true would not be complete without echoing the joy expressed in the historical section of this report over the grant by the American Philosophical Society of funds which will permit the library to embark on a project of cataloging its backlog of historical material.

Eberhard Foundation Grant. The Directors of the Eberhard Foundation approved the renewal of the grant which has made it possible for the library to subscribe to *Chemical Abstracts.* This gift is especially appreciated in these days of increasing awareness of the need for adequate bibliographical control of the literature.

B.W.L. Foundation. A grant was received from the B.W.L. Foundation through the kindness of Folz, Bard, Kamsler, Goodis & Greenfield. This money was allocated for the purchase of medical books.

Freshman Tours. The library was again given the opportunity of welcoming the Freshman medical students from Hahnemann Medical College, Temple University School of Medicine, and the Woman's Medical College. These groups were given brief talks on the history and functions of the College and were escorted on conducted tours through the building.

Personnel

Miss Josephine Walsh resigned from the position of Assistant Librarian, Readers' Service, in order to seek employment on the West Coast. Miss Walsh's responsibilities for the supervision of the Medical Documentation Service were assumed by Mr. Robert Blanken, the newly appointed Director of the Service. The reference and circulation aspects of this position were filled by the appointment of Mrs. Elnora Smith as Reference-Circulation Librarian. Mrs. Smith was previously employed as the Librarian of St. Joseph's Hospital in Denver.

The vacancy created by the resignation of

Mrs. Lillian Pellicano from the position of Assistant Cataloger was filled by the appointment of Miss Zena Babych. Miss Joyce Cushmore was appointed to the position of Readers' Service Division Assistant to fill the vacancy created by the resignation of Miss Ella Wallace. Two members of the present paging staff, Mr. Robert Glass and Mrs. Norma Picou have been appointed during the current report year. Personnel changes made since the end of the report year are indicated on the staff roster below.

Professional Staff Activity. The Medical Library Association is being served by library staff members in the following positions: Miss Muriel Hodge has been appointed to the position of Assistant Placement Officer; Mr. Elliott H. Morse is serving on the Board of Directors.

The Philadelphia Regional Group of the Medical Library Association appointed the following staff members to its committees, 1957/58: Muriel Zeldis, Chairman Program Committee; Muriel Hodge, Chairman Nominating Committee.

Miss Muriel Hodge has accepted the position of Meals Chairman for the forth-coming convention of the Special Libraries Association which will be held in Atlantic City. Mr. Morse is serving on the hospitality committee of the Special Libraries Council of Philadelphia and Vicinity.

The library was represented at the annual meeting of the Medical Library Association, which was held in Rochester, Minnesota, by Miss Muriel Hodge, Miss Josephine Walsh, Miss Muriel Zeldis, and Mr. Elliott H. Morse.

Mr. McDaniel's activities are noted in the historical portion of this report.

Benefactors. A list of the donors of books and other library materials appears in the statistical section of this report. Special grants and the support of the local medical schools is acknowledged above.

The library is pleased to be able to report the following institutional subscriptions to the support of the library made under the terms of the Contribution Plan:

*Air-Shields, Inc.
J. T. Baker Chemical Co.
Barnes, Dechert, Price, Myers & Rhoads
Bernstein & Bernstein
Borden Company—Chemical Division
Chew, Harvey & Thomas
E. I. du Pont de Nemours. Haskell Laboratory
E. I. du Pont de Nemours. Lavoisier Laboratory
Eaton Laboratories
Freedman, Landy & Lorry
General Electric Company. Missiles & Ordnance Systems Dept.
Gray & Rogers
*La Wall & Harrisson
Lee Ramsdell & Co.
Lemmon Pharmacal Co.
Lewis & Gilman, Inc.
MacAndrews & Forbes Company
*McNeil Laboratories, Inc.
*Merck Sharp & Dohme
Morgan, Lewis & Bockius
*National Drug Company
Oxy-Catalyst Inc.
Pennsylvania Railroad Company
Chas. Pfizer & Co., Inc.
*Quaker Chemical Products Corp.
Richter, Lord & Levy
Rittenhouse Book Store
Rohm & Haas Company
Samson Laboratories
*Smith, Kline & French Laboratories
E. R. Squibb & Sons
*Wallace Laboratories Division of Carter Products, Inc.
Warner-Lambert Laboratories
Wyanel Laboratories
*Wyeth Laboratories
Wyeth Laboratories—Marietta

E. H. M.

MEDICAL DOCUMENTATION SERVICE

ANNUAL REPORT, 1957–58

During the past fiscal year, the Medical Documentation Service enjoyed a further expansion of its activities and personnel and was placed on a firmer financial footing by bringing its rates more into line with necessary expenditures.

The acquisition of a full-time director in July 1958 and the addition of several part time employees in the fall gave the Service a necessary continuity and flexibility of staff. At the same time the new rates, which were approved

* Institutions which have made contributions in excess of Contribution Plan standards.

at a joint meeting of clients with officers of the College and members of the staff in November 1957, enabled the Service to support itself from current income. Mrs. Jean Stevens continues in the position of executive secretary, while Mr. Robert Blanken, who was previously employed on a part-time basis, assumed the post of director. The resignation of Miss Josephine Walsh, who had the responsibility for direction of the service during most of the fiscal year (until July, 1958), was a great loss.

The November meeting occurred shortly before the cessation of contracted bibliographical activities at the library of the New York Academy of Medicine, which resulted in a number of new clients for MDS from the New York area. With the approval of both new and old clients and the representatives of the College of Physicians, significantly increased institutional rates were established for the current-scanning, abstracting, and literature-searching activities of MDS, with a discount being made available to unaffiliated individuals and the original low rates being maintained for Fellows of the College. The increased income which was thus made possible enabled the Service to become self-supporting during the second half of the fiscal year.

The addition of a new type of service was made possible in July by the generosity of Wallace Laboratories, who made a microfilm camera available to MDS for both their own and other work. One of the uses to which this new facility has been put is to provide copy to Mr. Jerome Rauch of the New Jersey Academy of Medicine who is currently publishing a Tables of Contents Bulletin. Also, a number of journals have been filmed in their entirety for other libraries.

The library also entered the educational field in a small way in the fall with a course in Introductory Scientific Russian for librarians and technical translators, taught by the director of MDS. This course met with wide interest and enjoyed the participation of a number of members of the staffs of this and other libraries.

Meanwhile, the volume of regular activities expanded considerably during 1957/58, particularly the literature-searching operations of MDS. A number of new clients were obtained, some from states as far away as Iowa and Minnesota, emphasizing the position of MDS as the only service of its type attached to a major medical library.

The total income during the fiscal year 1957/58 was ca. $24,000, which was balanced by total expenditures of ca. $23,500. The major sources of income were the translation program ($14,000), the scanning service ($5,500), literature searching ($2000) and abstracting ($1000). A satisfying increase in all categories except translations was noted during the latter half of the year. During 1957/58, the Service reported 3996 references from the current literature on approximately 25 subjects, produced 361 abstracts and 187 translations (plus translations of 1871 Russian abstracts), and carried out 126 literature searches. The Service has also assisted in the writing of a number of scientific articles and has thus been of assistance to all categories of possible clients, from government agencies to pharmaceutical companies and from indexing services to individual physicians, research scientists and lawyers.

The increased use of MDS by clients of all types indicates its continued importance as an extension of the facilities of the College library, designed to make the resources of the library more available and more useful to its users.

R. R. B.

HISTORICAL

GENERAL

As the plangent voice of 'Historical' may be heard throughout the land on a fairly regular schedule, *via* its *Fugitive Leaves*, and in the compound, additionally, *via* regular reports to Library Committee and Council, an annual report from this quarter can represent little, if anything, more than a re-telling of twice-told tales. Yet it does serve usefully, in its way, as a sort of anthology of, and handy reference to, the more significant of the year's developments.

American Philosophical Society Grant. One of the major developments in the period covered by this report, certainly, was the College's receipt of a grant of $13,000 from the American Philosophical Society, for the purpose of enabling us to catalogue a large accumulation of historical materials. For the obtaining of this generous and most welcome aid to expansion of the usefulness of the historical collections, we are particularly indebted to our Past-President Dr. J. Parsons Schaeffer, our President, Dr. Jonathan E. Rhoads, our Fellows Dr. Esmond R. Long and Dr. Joseph Stokes, Jr.; and to Prof. E. Newton Harvey, of Princeton, and Miss Gertrude L. Annan, Librarian of the New York Academy of Medicine. Though official notice of the grant was received early in May, the search for an available cataloguer with the esoteric equipment indicated was so protracted (though not unexpectedly so) that no earlier a starting date for the project than 16 Feb. 1959 has been obtained.

Section on Medical History. Equally constructive, from the long view, and heart-warming in its familial implications, was the decision of the Executive Committee of the College's Section on Medical History to increase the Section's membership dues by two dollars, beginning in 1958, with the objective of donating the additional income towards expansion of the historical collections. During the year, accordingly, the collections benefited in the amount of $316 from this source—an indication of the appreciable enrichments of them that will be made, in the Section's name, as the years go on. Any public record of so significant an action in the library's interests again should allow us to associate it with its initiating angels—in this instance, Dr. Nicholas Padis, chairman at the time, and his co-members of the Executive Committee, Drs. Herbert J. Dietrich, Jr., Maurice S. Jacobs, Herbert T. Kelly, and Samuel X Radbill.

Acquisitions. Through gift or purchase, the historical collections were materially increased. Descriptions of a number of the acquisitions were furnished in the *Fugitive Leaves* (No. 17, Nov., 1957; No. 18, Dec., 1957; No. 21, April, 1958; No. 22, May, 1958); and of some others, in the *Reports of the Library Administration* (Dec., 1957; Jan., 1958; March, 1958; April, 1958; May/Aug., 1958; Sept., 1958; Oct., 1958). While the names of all donors appear under the heading 'Donations' elsewhere in the library's annual report, exception will not be taken, we believe, to our recording here also the names of a few whose continuous enrichment of the historical collections demands more than routine acknowledgement: Drs. Edward B. Krumbhaar, Samuel X Radbill, Fred B. Rogers, and J. Monroe Thorington.

Transfer Progress. It was noted in our last preceding report that the task of locating in the old stacks all material published before 1801 and transferring it to the rare-book floor of the new stacks had not been completed. It was completed during the past year, with the exception of the 'elephant' folios, which are being allowed to remain on the recumbent-elephant shelving judiciously created for them a half-century ago. There remains to be selected and transferred to the 'cage' (a residual term being retained for its monosyllabic convenience) material of the 19th and 20th centuries that falls into the 'rare-book' category.

FUGITIVE LEAVES

Seven numbers (N.S., Nos. 17–23) of the *Fugitive Leaves* were issued during the report year, as against nine in the last preceding year. Nos. 17 and 18, as noted above, were devoted to descriptions of some recent acquisitions; Nos. 19–21 presented a check-list of manuscript lecture notes in the library, the last including also descriptions of some further acquisitions; No. 22 was a miscellany of notes and description of an acquisition; No. 23 was the first installment of a check-list of 18th century American medical graduates of the University of Edinburgh, with the titles of their theses and supplementary information. There is some reason to believe that the citrus-colored (our unequivocal response to requests for a color distinguishable from that of the accompanying

Booklist) leaves continue to engage the interest of the library's friends even outside the family circle.

EXHIBITS

The "Historical Handbook" issued at the time of the Clinical Meeting of the American Medical Association in Philadelphia, December, 1957 (copies may still be obtained from the curator), shows that the library's historical collections were well represented at the historical exhibit in Convention Hall; and indicates that a supplementary exhibit was on view in the Hall of the College. The five meetings of the Section on Medical History were provided, as usual, with exhibits pertinent to the respective programs. The induction of the College's thirty-seventh President prompted an exhibit recalling the features and some publications of the distinguished physicians who had earlier occupied the chair. An historical exhibit, international in scope, was held in honor of the Fifth International Congress of Internal Medicine during its meeting in Philadelphia. The presentation, by Mrs. George B. Wood, of a handsome genealogical chart showing the relationship of doctors of medicine in the Wood-Bacon-Packard families gave occasion for an exhibit recalling the appearance and publications of some of the notable members of the families.

NON-ROUTINE ACTIVITIES

The curator read a paper entitled "A Brief Sketch of the Rise of American Medical Societies" before the Philadelphia Regional Group of the Medical Library Association; the paper was subsequently printed in *International Record of Medicine*, vol. 171, no. 8, August, 1958. For a class from the Drexel Institute of Technology he demonstrated some rare works in the history of nutrition. A note on "Harvey Presentation Copies", in the *Journal of the History of Medicine and Allied Sciences*, vol. 13, no. 4, Oct., 1958, sought to correct an impression he might earlier have fostered, that an indicated Harvey presentation copy in the library's collections was the only one of which there was record. He was appointed a member of the American Association for the History of Medicine's Osler Medal Committee for the year 1958/59.

W. B. McD., 2d

STATISTICAL SUMMARY

Inventory

Accessioned items[1]	*1958*		*1957*	
Incunabula[2]	419		419	
Manuscripts and typescripts	927		924	
General	194,588		191,066	
Accessioned pamphlets	4,415		4,380	
		200,349[3]		196,789

[1] Accessioned items in this library comprise the total number of bound volumes and such unbound pamphlets as have been catalogued as separate bibliographical units regardless of their size.

There remain many thousands of unaccessioned pamphlets awaiting accessioning and cataloguing. A true quantitative and qualitative picture of the library's holdings cannot be presented until this wealth of neglected material has been processed and duly registered in our 'accessioned', or 'volume', count.

[2] The library owns 421 incunabula but there are two cases of two bound together, reducing the number of accessioned items.

[3] Included in this figure are the following collections which are on permanent deposit in the library: Gross Library, 3,976 accessioned and 4 unaccessioned items; Parry Library of the Obstetrical Society of Philadelphia, 217; Mütter Museum, 200 (Mütter Museum books may not be removed from the College building).

Unaccessioned items[1]				
Reports	28,791			
Theses	59,521			
Pamphlets and reprints	231,453			
		319,765		318,832
Portraits[4]	102,403		101,333	

CURRENT SERIALS

	Subscription	*Gift*	*Exchange*	*Total*
U. S. A.	320	422	65	807
Western European Continent	602	62	112	776
Latin America and West Indies	15	75	75	165
United Kingdom and Eire	106	24	33	163
Canada	9	17	9	35
Eastern European Continent	19	17	20	56
Asia (including Japan)	15	46	45	106
Africa	2	5	13	20
Pacific (including Australasia)	2	9	10	21
United Nations	10	0	0	10
International	4	5	1	10
Grand Total: 1958	1,104	682	383	2,169
Grand Total: 1957	1,192	636	341	2,169

New titles: A total of 79 serials was added in 1957–58. 37 of these were purchased; 42 received in exchange or gratis.

Separate Issues received, checked-in on the cards, and shelved totaled 18,425. In addition, thousands of duplicate issues were received as usual. No attempt was made to count those held merely for exchange or other use.

Visitors in the Norris Room for the year totaled 3,311.

READERS' USE OF THE LIBRARY

Library Hours. The library was open, during the academic year, from 9:30 to 9:30 on Mondays and Wednesdays from 9:30 to 5:30 on the other weekdays. During June, July, August, and the first half of September the library was open from 1 to 9:30 on Tuesdays; from 9 to 5 on the other weekdays, except Saturdays, when it was closed. The following legal holidays were observed: Thanksgiving, Christmas and New Year's, Memorial, Independence, and Labor Days.

Visitors:

	Totals
1958:	12,119
1957:	11,479

Circulation:

	Totals:	*Intra-mural*[5]	*Extra-murial*[6]	*Books*	*Journals*
1958:	53,831	(40,838	12,993)	(9,769	44,062)
1957:	50,393	(38,085	12,308)	(10,469	39,924)

Inter-library loans: The library sent 7,980 (1957: 6,926) volumes on inter-library loan to 237 (1957: 170) libraries in 32 (1957: 31) States including Washington, D. C. and 5 Canadian provinces and has borrowed 10 (1957: 15) volumes from 6 (1957: 10) libraries.

Photostats and Cormac Sheets: 8,831 Photostat and Cormac prints were furnished on request (1957: 4,432).

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[4] Including 124 oil, and 268 other, framed portraits in the care of the Committee on College Collections. The count of portraits represents the number of entries on the cards in the portrait catalogue. The entries represent, in turn, both loose portraits and those in books, many of them, of course, being merely different reproductions of a single original.

[5] The number of volumes "consulted in the library" includes only those supplied on demand. Readers have access to the bound volumes of periodicals and reference works kept on the shelves in the Reading Room; the Fellows, and occasionally others, by special permission, have access to the book-stacks. There are, therefore, many volumes consulted of which no accurate record can be kept.

[6] This figure includes 1,084 unbound current issues circulated from the Periodicals Room.

Acquisitions and Accessions

Accessioned Acquisitions:

1958: 3,560 volumes (2,074 by purchase; 1,135 by gift; 351 by exchange). (*Books*: 976; *Periodicals*: 2,549; *Pamphlets*: 35).

1957: 4,110 volumes (2,364 by purchase; 1,367 by gift; 379 by exchange). (*Books*: 1,413; *Periodicals*: 2,662; *Pamphlets*: 35).

Non-accessioned Acquisitions:[7]

1958: 44 Reports (1957: 258)
854 Theses (1957: 512)
15 Miscellaneous items (1957: 259)

Donations

The donors for the past year number 127 (1957: 154). The library is indebted for large gifts of books, pamphlets, and unbound periodicals to the following donors: Children's Hospital; F. A. Davis Co.; Institute of the Pennsylvania Hospital; Jefferson Medical College Library; Drs. R. A. Kern, E. B. Krumbhaar; Lea & Febiger; J. B. Lippincott Co.; Merck Sharp & Dohme; Philadelphia College of Osteopathy; Dr. F. B. Rogers; W. B. Saunders Co.; Smith Kline & French Laboratories; Wistar Institute; Dr. I. J. Wolman; Woman's Hospital; Wyeth Laboratories.

Various publishing houses, among the large group of corporate donors, have presented volumes as follows: F. A. Davis Co., 9; Lea & Febiger, 8; J. B. Lippincott Co., 31; W. B. Saunders Co., 81.

Other individual donors were as follows: Dr. D. L. Baxter; Mrs. P. T. Bohan; Drs. W. N. Bradley, D. M. Davis; Dr. P. Drant Estate; Drs. G. G. Duncan, W. E. Ehrich, J. T. Freeman, B. C. Gettes, F. Guerra, H. J. Harwick, E. Hess, J. Johnson, T. S. Kirkbride, J. V. Klauder, H. Klickstein, D. W. Kramer, W. M. Krogman, C. H. LaClair, Jr.; Mr. W. R. LeFanu; Drs. E. R. Long, D. Macfarlan; in Memory of Dr. J. W. McConnell; Drs. T. G. Miller, S. W. Newmayer (through Dr. S. X Radbill), C. M. Norris, G. W. Norris, K. E. Paschkis, A. H. Persky, S. X Radbill, A. N. Richards; Mrs. L. Rosenthal; Drs. J. B. Rudolphy, L. C. Scheffey, H. Scheie; Mr. H. S. Schuman; Drs. E. Shaffer, W. B. Shelley, E. A. Strecker, J. M. Thorington, H. Tumen; Mrs. C. Urbach; Drs. T. E. User, F. J. Vossenberg, R. G. Williams, F. C. Wood; Miss M. Zentmayer.

The *Transactions & Studies* was sent, either in exchange or as gift, to 455 organizations.

The library received a number of journal issues through gifts and supplied journals to other libraries through a variety of channels.

Theses and dissertations to the number of 854 have been received on exchange from the following European schools of medicine: Universities of Basel, Bern, Copenhagen, Erlangen, Lausanne, Louvain, Lund, Paris, Uppsala, Utrecht.

The Staff[8]

Elliott H. Morse	*Librarian*
H. Muriel Hodge	*Assistant Librarian*
Zena Babych	*Cataloging Assistant*
Joyce Cushmore	*Readers' Service Assistant*
Robert Glass	*Library Page*
Catherine Ledwell	*Library Page*
Mrs. Kathryn H. Miragliotta	*Order Librarian and Cataloging Assistant*
Dorothy Minton	*Serials Assistant*
Dolores Pagano	*Library Secretary*
Mrs. Norma Picou	*Library Page*
Mrs. Elnora Smith	*Reference-Circulation Librarian*
Robert Souza	*Library Page*
Muriel Zeldis	*Serials Librarian*
W. B. McDaniel, 2d	*Curator of the Library's Historical Collections*
Mrs. Maria Shchuka	*Rare Book Cataloger*
Robert Blanken	Medical Documentation Service, *Director*
Mrs. Jean C. Stevens	Medical Documentation Service, *Executive Secretary*

Approved:

John Franklin Huber, M.D.
Chairman

[7] As usual, not all the pamphlets, theses, and numbers of various journals listed as received had been checked for keeping or discard at the close of the library year.

[8] Library staff list as of February 16, 1959.

Annual Report of the Committee on the Mütter Museum and College Collections

THE Mütter Museum of the College of Physicians was open daily except Saturdays and Sundays, from 9.30 A.M. to 1.00 P.M. and from 2 P.M. to 4.30 P.M. and until 8.30 P.M. on Stated Meeting Nights of the College. It was closed during August due to vacation.

This year 1895 visitors inspected the various exhibits in the Museum. Besides the many individuals from this country and from foreign countries, there were large classes from the Medical School of Temple University, Hahnemann Medical College, Woman's Medical College and the Franklin School of Science and Arts. Students from the Girls' High School who are interested in nursing and student nurses from Montgomery Hospital in Norristown also found their visits rewarding.

As in previous years Dr. Samuel X Radbill's class in Pediatrics spent a profitable afternoon in the Museum.

On December 11, 1957, Dr. R. Philip Custer delivered the Seventieth Thomas Dent Mütter Lecture, the title being, *The So-called Hypersplenic Syndrome; It's Pathogenesis, Recognition and Management.* On that same evening the present and former members of the Committees on the Mütter Museum honored the curator, Mrs. Ella N. Wade, for her eighteen years of service in the Museum, by presenting her with a handsome carafe and tray suitably engraved for the occasion.

The Seventy-first Thomas Dent Mütter Lecture was delivered October 1st, 1958, by Dr. Peter A. Herbut on *Studies on the Properdin System in Transplantable Cancer.* At the dinner preceding the lecture Dr. Herbut was presented with an engraved mug as a memorial of the lecture, by the members of the Mütter Museum Committee.

The following gifts were accepted as permanent exhibits in the Museum:

Presented by Dr. M. Luther Kauffman:

An unusual pair of tinted spectacles

Presented by Dr. N. William Winkelman, Jr.:

Mallet and calipers used by his father, Dr. Nathaniel W. Winkelman, to perform a transorbital lobotomy.

Mrs. Ella N. Wade was reappointed curator for the year 1958–59.

College Collections

Mr. Frank Ireland restored three portraits during the year, namely those of Dr. Frederick Packard, Dr. Robley Dunglison and Dr. Benjamin Rush.

The committee furnished Mr. Herman Friis, Chief Cartographer, Record Division, National Archives, Washington, D. C., with a photograph of the College portrait of Alexander von Humboldt by Charles Willson Peale. Dr. Frank P. Albright, Director of Museums, Old Salem, Winston-Salem, North Carolina, was given a photograph of the portrait of John Hunter by Charles Robert Leslie after Sir Joshua Reynolds.

The following gifts have been added to College Collections:

Presented by Dr. Walter B. Shelley from the estate of Dr. Weidman:

Three medals:

Lucien Marie Pautries
Theobald Smith
Raimond Jacques Sabouraud

Presented by Dr. T. Grier Miller:

The American Gastro-Enterology Association medal, picturing Julius Friedenwald, awarded Dr. Miller in 1950 for outstanding achievement in gastroenterology.

Presented by Dr. Joseph Ritter:

Medal of the Philadelphia Pediatric Society awarded Dr. Howard Childs Carpenter.

Presented by Dr. S. Dana Weeder:

Microscope formerly belonging to Dr. W. W. Keen.

Presented by Dr. Jonathan E. Rhoads and Dr. Lewis C. Scheffey:

Oil portrait of Dr. Robley Dunglison by Thomas Sully.

Presented by Dr. Edward B. Krumbhaar:

Large English mortar and pestle formerly used by Dr. Conyers Middleton (1683-1750) in his chemical experiments.

Large wooden mortar and pestle from Concord, Massachusetts, 18th century, of local manufacture and use.

Iron press for making suppositories.

Presented by Dr. Catharine Macfarlane:

Radium applicator used by Dr. Macfarlane in treating epithelioma of the vulva in 1903, the first time radium was used in Philadelphia.

Presented by Dr. J. Monroe Thorington:

Six silver votive offerings commonly used in the Greek Orthodox churches of Athens.

Five silver votive offerings from Kos.

Mortar and pestle from Tyrol, Austria, 1790.

Six artificial eyes of shell and blown type

One glass and four gold Prostheses for implantation after enucleation, that formerly belonged to Dr. James Thorington.

Fifteen Roman and Greek coins with medical significance, namely:

Epidaurus 323–240 B.C. (Two)
Pergamum/Mysia ca. 2nd century B.C.
Acilius ca. 55 B.C.
Hierapolis/Phrygia Nero, 54–68 A.D.
Hadrian 117–138 A.D. (Two)
Marcus Aurelius 161–180 A.D.
Caracalla 211–217 A.D. (Two)
Maximinus 235–238 A.D.
Trebonianus Gallus 251–253 A.D. (Two)
Valerian 253–259 A.D.
Postumus 258–267 A.D.

Presented by Dr. Douglas Macfarlan:

Esophageal stethoscope for use in chest operations.

Presented by Mr. Leland Rosemond through Dr. Douglas Macfarlan:

Otarion Listener, an eye-glass hearing aid.

Presented by Dr. Burton Chance through the Wills Eye Hospital:

Dr. Thomson's lantern for detecting color blindness for railway and marine service.

Presented by Dr. Douglas P. Murphy:

Six models of infants' heads showing distortion of the head due to the difference in presentation at birth.

Presented by Miss Katherine McConnell:

Clinical thermometer formerly belonging to Dr. J. W. McConnell.

Presented by Mrs. Alice E. Hart:

A microscope at least one hundred years old that formerly belonged to Dr. Charles Y. Swan who practiced in New York.

Presented by Dr. Samuel X Radbill:

Six Roman and Greek coins with medical significance, namely:

Antoninus Pius 138–161 A.D.
Nikaia/Bithynia, Marcus Aurelius 161–180 A.D.
Crispina, wife of Commodus. Died ca. 183 A.D. (Two)
Serdica/Thrace, Caracalla 211–217 A.D.
Marcianopolis/Moesia, Elagabal 218–222 A.D.

Presented by Miss Mary Zentmayer, a bequest from the late Dr. William Zentmayer:

Four medals awarded Joseph Zentmayer for his work on microscopes:

Bronze medal awarded by the United States Centennial Commission in 1876.

Silver medal awarded by the Paris Exposition, Universelle Internationale in 1878.

Silver medal awarded by the Franklin Institute in 1874.

Elliott Cresson medal (founded in 1848) awarded by the Franklin Institute in 1875.

Two medals awarded Dr. William Zentmayer:

Leslie Dana medal awarded in 1945 for prevention of blindness.

Lucien Howe medal of the American Ophthalmological Society for distinguished service to ophthalmology.

Presented by the Wistar Institute:

Plaster bust of Caspar Wistar, one of four

copies of the original made by the sculptor, William Rush.

Presented by Mr. Asger F. Langlykke of E. R. Squibb & Sons:

Centennial medal of E. R. Squibb & Sons.

Presented by Dr. Herman Beerman:

Hypodermic syringe, date 1897.

Dr. Harry Shubin presented two instruments that were duplicates of some in the Museum. With Dr. Shubin's permission these were kept to exchange with other museums.

Presented by Mrs. Alfred Stengel:

Academic robe formerly belonging to Dr. Alfred Stengel.

Mrs. Ella N. Wade was reappointed custodian of College Collections for the year 1958–59.

THEODORE F. BACH, M.D., *Chairman*

Memoir of Elliston Joseph Morris (1862–1958)*

By JOHN H. ARNETT, M.D.

ELLISTON JOSEPH MORRIS, born in Philadelphia, of Galloway Cheston and Hannah Perot Morris, died in Devon, Pa., on August 31, 1958, at the age of 95. He prepared for college in the Germantown Academy, graduated from the University of Pennsylvania in 1883, and attended its Medical School during Dr. Osler's inumbency. He was a member of the Phi Kappa Sigma Undergraduate Fraternity, and, after graduating from the Medical School in 1886, he received the degree of A.M. from the University of Pennsylvania.

Since his grandfather, Dr. Caspar Morris, had been one of the founders of the Episcopal Hospital, it was here that young Elliston cast his lot, serving first as Resident Physician and then as Visiting Physician to the Out-Patient Department. In 1897, he was appointed Visiting Physician to the Hospital, and in 1921 Chief of Medical Service "A", which position he held until December 1st, 1926, when, after 40 years of active service to the Hospital, he retired with the rank of Consulting Physician, and, later, Emeritus Chief.

In 1889, when the news of the Johnstown flood reached Philadelphia, he volunteered for Red Cross relief work in the ruined city and worked "with little sleep and less to eat" until the chaos of mud, water and wreckage gave way to some semblance of order. The dead having been washed and buried, the sick and injured cared for in improvised hospitals, the hungry fed, the wreckage blasted and burned, the relief party then returned to Philadelphia.†

Doctor Morris for many years was employed as a Company Physician by both the Girard Trust Company and the Baltimore and Ohio Railway, and during World War I he was a member of the Volunteer Medical Service Corps, and the Advisory Board of the Episcopal Hospital.

He was active in the affairs of the College of Physicians of Philadelphia from the time of his election in 1893 until he retired from active practice, serving as Elective Councillor from 1901 to 1903, Chairman of the Alvarenga Prize Committee in 1905, and on the Hall Committee from 1923 to 1933, being Chairman of this Committee in 1933.

He was a member of the Obstetrical Society of Philadelphia, became its Secretary in 1892, and upon his retirement was made an Honorary Member. He was also a member of the Medical Club, the Philadelphia County and Pennsylvania State Medical Societies, and an Associate member of the American Medical Association.

In 1892, he married Elise C. Heydecker. After her death, he married Josephine C. Le Moyne in 1908. He is survived by two sons, Elliston J., Jr., and Edward Heydecker.

During his many years of practice he lived at 128 South 18th Street. Cheerful and genial with all who knew him, he was a physician of the old school, who took a personal as well as professional interest in every one of his patients.

* Prepared and published at the request of the Council of the College of Physicians of Philadelphia.

† This work, under the direction of Clara Barton, founder of the American Red Cross, is described in a letter from Dr. Morris to Alice Graham Lanigan, which is the property of this College.

Memoir of William Zentmayer (1864–1958)*

By WARREN S. REESE, M.D.

DOCTOR WILLIAM ZENTMAYER, one of the most active and prominent members of this Section, died on the evening of March 18, 1958, in his 94th year, during an attack of angina pectoris. This attack was secondary to a coronary thrombosis suffered one month earlier. Thus passed a distinguished, possibly unique figure in ophthalmology—a man who, during his active career, was one of a galaxy of prominent ophthalmologists, many of whom graced the membership of this Section.

Doctor Zentmayer was born October 28, 1864, the son of Joseph and Katherine Zentmayer. His father, who emigrated from Germany, was an eminent optician and he undoubtedly influenced his son to study ophthalmology. Mr. Zentmayer established his firm in Philadelphia in 1853 and soon won world-wide renown for his work and inventions on microscopes. The Franklin Institute awarded him the Elliott Cresson gold medal in 1874, the Centennial Exhibition in Philadelphia a second medal in 1876, and the Paris Exhibition a third medal in 1878 for his work on microscopes. During this time, there was not a maker of microscopes in the world who did not use some of his important inventions. A photographic lens that he conceived theoretically, and then made, was so radically original, that when it was first described, his European confreres called it incredible and an "American exaggeration". This caused a controversy which is detailed in various issues of The Journal of the Franklin Institute and The Philadelphia Photographer. He was among the first to limit his business in spectacles to prescriptions from ophthalmologists; and as early as 1860, ground sphero-cylindrical lenses. It was due to men like Doctor Zentmayer and his father, that during this period, Philadelphia was one of the greatest ophthalmological and optical centers in the world.

Doctor Zentmayer's numerous contributions to literature were all carefully and thoughtfully executed, and he was frequently asked to discuss papers because of his knowledge of ophthalmic literature. He was Chairman of the Section on Ophthalmology of the American Medical Association 1916–17, and President of the American Ophthalmological Society, 1926–27. These positions and others he obtained through sheer merit, as he was not especially gregarious though a witty and interesting conversationalist. He was a bachelor, and his main interests were ophthalmology, poetry and music. He was an insatiable reader and had a remarkably retentive memory for things ophthalmological, so that his knowledge of ophthalmic literature was second to none, as many an essayist can testify.

Doctor Zentmayer was a good surgeon, and performed a greater variety of operations than any one I know, particularly on the extraocular muscles. He was an excellent teacher and his lecture notes were in great demand. In 1919, he helped organize the basic training course in ophthalmology in the Graduate School of Medicine of the University of Pennsylvania. During his active career, he was considered "the oracle" at Wills Eye Hospital, where all sorts of questions were put to him. If he was unable to answer a question at the moment, one could be sure that he would have a very full explanation within a day or so, having consulted his well-stocked library in the meantime. His editorial training made him very critical and I know of no one who so frequently discovered ophthalmology spelled incorrectly. He was a keen clinician and remembered his patients remarkably, and woe betide the resident who forgot his orders or findings of the previous clinic day. His forte, however, was ophthalmoscopy and, curled up on a stool in

* Read before the Section on Ophthalmology, College of Physicians of Philadelphia, 23 October 1958.

the dark room of the old Wills Hospital, he astounded students and ophthalmologists alike by his ability to determine the refraction of an eye with his insignificant-looking little reflecting light ophthalmoscope.

Doctor Zentmayer served as Editor of the Archives of Ophthalmology from 1950, when it was taken over by the American Medical Association, up to the time of his death, having previously served as Associate Editor. He enjoyed the friendship and respect of Doctor Arnold Knapp, who had edited and published this periodical at great expense to himself prior to this transfer. Doctor Zentmayer also served as editor of The Transaction of the College of Physicians of Philadelphia from 1899 to 1913, and was very proud of his portrait by Daniel Garber, presented to that institution by an international group of ophthalmologists in 1940. He was President of the Medical Alumni Society of the University of Pennsylvania in 1922–23, and was guest of honor at the Annual Meeting of the American Medical Association in 1941.

The St. Louis Society for the Blind awarded him the Leslie Dana Medal in 1945 for "Outstanding Achievements in the Prevention of Blindness and the Conservation of Vision"; and in 1948, he was awarded the Lucien Howe Medal by the American Ophthalmological Society "In Recognition of His Distinguished Service to Ophthalmology".

Doctor Zentmayer began his career as Chief of the Eye, Ear, Nose and Throat Out-patient Department of the Children's Hospital in 1887. He also served at the Glen Mills School, St. Mary's Hospital, St. Agnes Hospital and The Wills Eye Hospital, where he became Attending Surgeon in 1901. He resigned this position at age 65 of his own volition and retired from active practice on February 2, 1951—largely, I believe, because of a macular choroiditis. Fortunately, he was able to read with one eye up to the time of his death, and thus continue his interest in ophthalmology.

Doctor Zentmayer was very regular in his habits and punctilious in the amenities of his scientific and social life. During his active career, he smoked two cigarettes daily, one when he finished his clinic and one after dinner. He rarely, if ever, missed a Section Meeting of the College of Physicians of Philadelphia and I doubt if he missed many meetings of the American Ophthalmological Society. He often decried the present day need of guest speakers at this Section. He walked daily and knew all of the walking paths at Hot Springs, Virginia, and his favorite Summer resort, Lake Minnewaska, where he would cover as much as 15 miles over mountainous terrain, hopping along like a bird. As a younger man, he bicycled and was very proud of his "Century", or 100 mile ride from Philadelphia to Atlantic City and return on a warm Summer day, and particularly of achieving this without wilting his high starched wing collar! Indeed, he belied the oft-heard athletic adage that the "legs go first". I doubt if he enjoyed anything more than the various ophthalmological meetings, especially those of this Section and the American Ophthalmological Society, and one could fill a book with his numerous and interesting anecdotes of the various members.

At the time of his death, he was consulting surgeon to the Wills Eye Hospital, Emeritus Professor of Ophthalmology at the Graduate School of the University of Pennsylvania, Editor of the Archives of Ophthalmology, and a life member of the National Society for the Prevention of Blindness. He lived at 265 Forrest Road, Merion Station, Pa., and is survived by a sister, Mary.

Now Mr. Chairman, with your permission, I would like to present through this Section, certain articles that Doctor Zentmayer has bequeathed to the College. These include the medals which I mentioned in the memoir that were given to Doctor Zentmayer and his father; two volumes entitled "A View of the Causes and Progress of the French Revolution", by Dr. John Moore, printed in 1795 in Dublin, and a volume presented to Doctor Zentmayer by the Graduate School, Class of 1938–1939, comprising his lecture notes on "The Theory and Practice of Ophthalmoscopy".

Memoir of Walter Roberts (1870–1958)*

By GEORGE M. COATES, M.D.

WALTER ROBERTS, B.S., M.D., was born in Moorestown, New Jersey, on May 13, 1870, and died March 21, 1958, at the age of eighty-eight. His parents were Emmor and Martha Lippincott Roberts, both from old New Jersey families, members of the Society of Friends, of which Walter was, therefore, a birthright member, and in which Faith he remained throughout all his days.

His early education was in the local schools of Moorestown, and his collegiate years were spent at Swarthmore College, where he obtained the degree of Bachelor of Science in 1890. While a collegiate undergraduate he was a noted athlete, and, although small in size, probably the greatest track man ever produced at this institution. In his senior year he won five championships in the Intercollegiate Athletic Association of Pennsylvania meet. Throughout his medical study years at the Medical School of the University of Pennsylvania he continued his physical training and took part in intramural contests, being no longer eligible for intercollegiate competition. He was an expert croquet player, and played badminton until 1951.

He graduated in medicine in 1893, and served a two-year internship, then called a residency, at the Pennsylvania Hospital. It was at this time that he took up golf, which he continued even into his advanced years, and in which he won a number of championships of the Springhaven Golf Club, of which he was a member.

Dr. Roberts began the study of otolaryngology, of which he was to become a master, at the conclusion of his internship, on the services of Dr. George Harlan and Dr. Alexander McCoy at the Pennsylvania Hospital. Soon thereafter he joined the staff of the Methodist Hospital, later becoming Chief of Service in Otolaryngology, a position he held for many years. He joined the staff of the Philadelphia Polyclinic and College for Graduates in Medicine, and went from Assistant in Otology through Associate to Professor of Otology in about 1915. In the meanwhile he had been appointed Chief of Otolaryngology in the Philadelphia General Hospital, which position he also held with credit for a long period.

During the first World War he served on the Medical Examining Board, and in 1917 he became Professor of Otology in the Graduate School of Medicine of the University of Pennsylvania, which resulted from the merger of the Polyclinic and the Medico-Chirurgical College. He begame Emeritus Professor and consultant in 1953, when his health was beginning to fail.

As a teacher of otology, his forte was in the clinic and dispensary, and he gave invaluable practical instruction to the graduate students. His knowledge of otolaryngology was profound, and his reading extensive, though he wrote very little. He was a skillful and sound surgeon, and a most helpful consultant. In the lecture room he was informal and practical, and he was very popular with his classes.

He was a delightful companion, very modest, with a good sense of humor, and one of the kindest and gentlest of men. He had the loyalty and affection of all of his staff members and colleagues.

He was a member of the Union League and the Springhaven Golf Club. He was a Trustee of Swarthmore College.

Dr. Roberts was married in 1900 to Lydia Williams, who pre-deceased him by a few years. He is survived by three children: Anna Brosius, Lydia Dunham and Gilbert, presently a Commander in the U. S. Navy.

In 1930, while touring Europe with Mrs. Roberts, they were much attracted by the

* Prepared and published at the request of the Council of the College of Physicians of Philadelphia.

island of Majorca in the Mediterranean, and on their return home purchased a villa there, where they spent pleasant summer vacations until the second World War put an end to the adventure.

Dr. Roberts became a Fellow of the College of Physicians of Philadelphia in 1899, and served for two years as Chairman of the Section on Otology and Laryngology. He was elected a Fellow of the American Laryngological, Rhinological and Otological Society in 1905, and of the American Otological Society in 1910, and was a Senior Fellow in each at the time of his death.

Memoir of Gabriel Tucker (1880–1958)*

By CHEVALIER L. JACKSON, M.D.

GABRIEL TUCKER was born in Wheeling, West Virginia, son of Thomas Fleming and Johanna Roach Tucker, on November 3rd, 1880. He graduated from the West Virginia State Normal School and subsequently attended the College of Physicians and Surgeons in Baltimore (1901-1903) He next attended the Jefferson Medical College from 1903 to 1905, when he received his medical degree.

Dr. Tucker interned at the Mercy Hospital in Pittsburgh and subsequently became an Associate in Surgery at that institution (1906–1908). He practiced industrial medicine and surgery, and then otolaryngology, in the Pittsburgh area for ten years. In 1918 he served in the Medical Corps of the Army, at Base Hospital #126, with the rank of Captain.

In 1919, Dr. Tucker became associated with Dr. Chevalier Jackson, first at Jefferson and subsequently at the University and Graduate Hospitals. In 1930 he succeeded Dr. Jackson as Head of the "Bronchoscopic Clinic" at the University and Graduate, becoming Professor of Bronchology and Esophagology. This title at the Graduate School was changed to Professor of Bronchoesophagology and Laryngeal Surgery in 1932. In 1950 he was named Chairman of the Department of Bronchology, Esophagology and Laryngeal Surgery. He became Emeritus in 1954 and retired from practice the following year. Death came suddenly three years later, on April 17th, 1958.

During his entire professional life, Dr. Tucker was devoted to both peroral endoscopy and laryngeal surgery. He collaborated in postgraduate courses given at the Graduate School of Medicine and in Paris. When Dr. Jackson retired from the University of Pennsylvania to establish a clinic at Temple University, Dr. Tucker succeeded him and continued both the clinical work and the postgraduate teaching at the University hospitals, and in the Graduate School of Medicine. He devoted himself with undivided zeal to his professional work, having few if any hobbies or avocations. His genial personality made him many friends; he was at all times abstemious in his habits.

In 1937, Dr. Tucker was Guest of Honor at the meeting of the British Medical Association at Oxford, and there showed the first motion pictures of the larynx in color. He was a member of many local and national as well as several foreign medical societies. In 1932 he was Chairman of the Section of Otolaryngology of the American Medical Association. He was President of the American Bronchoscopic Society (now the American Broncho-Esophagological Association) in 1941. In 1944 he was President of the Philadelphia Laryngological Society and in 1945 Chairman of the Section on Otolaryngology of the College of Physicians of Philadelphia. He was President of the Laennec Society of Philadelphia and also a member of the John Morgan Society. Membership in foreign societies included the Scottish Society of Otology and Laryngology and the Société des Laryngologistes des Hôpitaux de Paris.

Dr. Tucker was actively interested in the development of bronchoesophagology, and established "bronchoscopic clinics" at the Misericordia (1924), Bryn Mawr (1927), Mt. Sinai (1929), Abington (1931) and Fitzgerald-Mercy Hospitals. He was consultant to the Woman's Medical College Hospital, the Jeannes Hospital and the Norristown State Hospital; he served for a time as assistant in laryngology at the Philadelphia General Hospital (1920 to 1924).

In the field of bronchoesophagology, Dr. Tucker invented numerous instruments for use in the removal of foreign bodies from the air

* Read before the College of Physicians of Philadelphia, 4 February 1959. Prepared and published at the request of the Council of the College of Physicians of Philadelphia.

and food passages, for example, the Tucker tack and pin forceps. He devised a retrograde bougie for use in the treatment of lye strictures and a valvular tracheal cannula for use especially in patients with bilateral laryngeal paralysis. An aspect of laryngeal surgery which particularly interested him and to the development of which he contributed much, was the surgical treatment of cancer of the larynx. His contributions to the literature of this subject were numerous.

Dr. Tucker was married in 1911 to Irene Jarvis, who died in 1918; and in 1923 to Beatrice Jarvis, who has survived him. Dr. Tucker is survived also by his three sons, Gabriel Tucker Jr., born in 1924, who is now head of the Department of Bronchoesophagology at The Johns Hopkins Hospital, Chevalier Jackson Tucker, born in 1926, who is advertising manager of the Chemical Division of Merck-Sharp & Dohme, and John Anwyl Tucker, born in 1930, who has just completed his internship at the Lankenau Hospital and is now a Resident in Otolaryngology and Bronchoesophagology at the Hospital of the University of Pennsylvania.

Memoir of Norris Wistar Vaux (1881–1958)*

By WILLIAM D. STROUD, M.D.

DR. NORRIS WISTAR VAUX was born on September 1, 1881, at Rosemount, Pennsylvania, the son of Jacob Waln Vaux, and Emily Norris Pepper Vaux. His popularity as a youth was evidenced by his election to the F.X.I. Fraternity, while at the Delancey School. At that time, this was the outstanding Schoolboy Fraternity among the Interacademic Schools. Dr. Vaux was graduated from the University of Pennsylvania School of Medicine in the year 1905. In his Senior year, he was elected to the Sphinx Senior Society, as one of the twenty five outstanding members of his Class. He was a member of the Fraternity of Delta Psi, and in addition to his many other accomplishments, rowed on the Varsity Crew. In the year 1901, Dr. Vaux enlisted in the First Troop Philadelphia City Calvalry and was elected Honorary Surgeon in 1911.

From the year 1905 through 1907, Dr. Vaux served a rotating internship at the Pennsylvania Hospital. His interest in obstetrics and gynecology developed there, and upon completing his internship, he traveled to Dublin, Ireland, with his bride Honora A. Dixon, to spend a year of post-graduate work in this specialty at the Rotunda Hospital.

Upon returning from Ireland, Dr. Vaux entered general practice in Chestnut Hill and Philadelphia. His great interest in medical students, and his exceptional teaching ability, soon became apparent to his associates, so that with Dr. Edmund Piper, he became Co-Chief of Obstetrics at the Philadelphia Lying-In and Maternity Hospital, then located at Eleventh and Cherry Streets. He became a Fellow of the College of Physicians of Philadelphia on January 3, 1912.

* Read before the College of Physicians of Philadelphia 7 January 1959. Prepared and published at the request of the Council of the College of Physicians of Philadelphia.

Everyone who knew Norris Vaux, loved and admired him as a man and a friend. He was the ideal physician—kindly, with a wonderful sense of humor, and always efficient and conscientious in his work. He was the type of doctor who would visit the newly delivered mothers on the Ward, the last thing in the evening, just to be sure that everything was all right, before going home.

His work was interrupted in 1917 when World War I began. He immediately enlisted in the Medical Corps and served with distinction for the next twenty three months. His tour of duty included the American Expeditionary Force—Base Hospital #10 (The Pennsylvania Hospital Unit) and the British Expeditionary Force—Base Hospital #16. At the end of the War, he was discharged with the rank of Major.

In 1919, Dr. Vaux resumed his practice and in 1925 was elected to the teaching staff of Jefferson Medical College. There he was made Professor of Obstetrics in 1936, a position which he held until 1947, when he was made Emeritus Professor.

In 1938, the Philadelphia Lying-In and Maternity Hospital merged with the Pennsylvania Hospital and Dr. Vaux became Director of the Department of Obstetrics and Gynecology of that hospital. He held this position until his retirement in 1946.

Having retired from active practice and teaching—in January, 1947—Dr. Vaux was appointed Secretary of Health of the Commonwealth of Pennsylvania, in which position he served until 1951.

Dr. Vaux contributed a total of twenty medical articles to various well recognized Medical Journals and Textbooks. He was the first Chairman of the Section of Obstetrics and Gynecology of the Pennsylvania State Medical Society in 1936 and President of the American Gynecological Society in 1946 and 1947—one

of the highest honors in American Gynecology and Obstetrics. During this year, the Senior Class of Jefferson Medical College presented his portrait to the College.

During the years between 1943 and 1947, Dr. Vaux served on the American Board of Obstetrics and Gynecology. He was a member of our Philadelphia College of Physicians; the Philadelphia Obstetrical Society; the American Committee on Maternal Welfare; the American Medical Association; the Philadelphia County Medical Society; an Honorary Member of the Barton C. Hirst Obstetrical Society and an Honorary Member of the Washington Gynecological Society.

His social club was the Philadelphia Club.

At a meeting of the Executive Committee of the Staff of the Bryn Mawr Hospital, held shortly following his death on August 19, 1958, the minutes record, "Dr. Vaux was one of the most distinguished physicians in the Philadelphia area. He served as Head of the Maternity Department and later as Consulting Obstetrician and Gynecologist from the year 1923 until the time of his death".

It is hard to believe that Norris Vaux has left us. His was a life of devotion to his patients and his profession. He is sadly missed by his many devoted friends.

Memoir of Albert E. Bothe (1891–1955)*

By FRANK B. LYNCH, JR., M.D.

ALBERT E. BOTHE was born in Camden, New Jersey on August 5, 1891. His parents were Alfred and Emma Catherine Bothe.

After finishing grade school Al, as his friends knew him, attended Pemberton Seminary, completing his course there in 1913. He then entered the University of Michigan, from which he was graduated in 1917.

Dr. Bothe received his Doctor of Medicine degree from the Long Island Medical College in 1920. He interned at the Long Island College Hospital and then returned to Philadelphia where he served a residency in pathology at the William Pepper Laboratory of Clinical Medicine at the University of Pennsylvania. In addition to his previous degrees, Dr. Bothe subsequently received the degrees of M.Sc. and D.Sc. from the University of Pennsylvania.

One of his many important early contributions was his organization of the Department of Surgical Pathology in the Department of Surgery at The Hospital of the University of Pennsylvania, which department he ably conducted for many years.

Hospital and teaching connections of Dr. Bothe included Associate in Urology at the Medical School of the University of Pennsylvania, and Professor of Clinical Urology at the Graduate School of Medicine of the same institution. He was Chief of the Department of Urology at Misericordia, Fitzgerald-Mercy and Jeannes Hospitals, and Consultant in Urology to the Children's Hospital of Philadelphia. While in the Navy during World War II, he became Chief of the Department of Urology at Bethesda Naval Hospital. He later held an Honorary Chair in the University of Madrid where he was a Lecturer in Urology in 1954.

Dr. Bothe was a member of the American Urological Association, the Eastern Surgical Society, the Philadelphia Academy of Surgery, the Middle Atlantic Urological Association, and the Philadelphia Urological Society (of which he was a Past President). He was elected a Fellow of the College of Physicians of Philadelphia on May 7, 1928 and was active in its affairs.

Dr. Bothe was an accomplished surgical pathologist, a leader in his chosen specialty of urology, and a prolific writer on subjects relating to bacteriology, surgical pathology and urology.

In 1922, Albert married Marion Bradley of Camden, New Jersey. They had one daughter, Anne, a beautiful and talented girl whose untimely death in 1951 was a shock from which her father never completely recovered.

Albert E. Bothe died on November 11, 1955, as the result of a cerebrovascular accident following a very short illness. Besides his widow, he is survived by one brother, Frederick A. Bothe, M.D. of this City.

* Prepared and published at the request of the Council of the College of Physicians of Philadelphia.

Memoir of Winifred Bayard Stewart (1899–1957)*

By CHARLES RUPP, M.D.

WINIFRED BAYARD STEWART, professor of neurology in the Woman's Medical College, died August 20, 1957 after a long illness.

Born in Philadelphia December 31, 1899, Dr. Stewart received her B.A. from the University of Pennsylvania in 1922 and M.A. in 1924. She graduated from the University of Pennsylvania Medical School in 1926 and then began her long association with the Philadelphia General Hospital where she served as an intern from 1926–1928. Shortly after completion of her internship, she married the late Dr. William A. Drayton, a well-known Philadelphia psychiatrist and Fellow of this College.

Dr. Stewart became associated with the neurologic group at the Graduate Hospital under Dr. Weisenburg and for many years was an associate in neurology at the Graduate School of Medicine of the University of Pennsylvania. She served as an assistant neurologist at the Philadelphia General Hospital from 1928 to 1938 in which year she became a chief in the psychiatric department. Eventually she became chairman of this department, a position she held at the time of her death.

Early in her career Dr. Stewart joined the psychiatric department of the Municipal Court of Philadelphia and in 1951 became the first director of the neuropsychiatric department of the Quarter Sessions Court. In these capacities it was her duty to examine psychiatrically many offenders against the law. She developed a unique facility in arriving at the core of the problem in a comparatively short time. She had no patience with the many "isms" and nebulous psychiatric theories. Her opinions, clear, concise, and almost invariably correct, commanded the respect of judges and attorneys; yet she was always sympathetic if the person were actually mentally ill.

In spite of her heavy clinical and teaching duties, Dr. Stewart always maintained a vigorous interest in neurophysiological research and in association with Drs. Grayson P. McCouch, and Joseph Hughes made a number of significant contributions to the literature.

In addition to being a Fellow of this College, she belonged to the Philadelphia County Medical Society, the Pennsylvania State Medical Society, and the American Medical Association. She was a Fellow of the American Academy of Neurology, the American Association for the Advancement of Science, and the Philadelphia Neurological Society of which she was at one time Vice-President. She was also a member of Sigma Xi and Alpha Omega Alpha.

Following the death of Dr. Drayton in 1952, Dr. Stewart married Norris S. Barrett, an attorney, who survives her.

Winifred Stewart was a staunch and loyal supporter of those causes which she thought right. Probably her greatest loyalty was to the Philadelphia General Hospital which she served so long. In all of the perennial instances where the autonomy of Philadelphia General Hospital was threatened by the various schemes of those who sought to manipulate the internal affairs of the hospital to their own advantage, she was in the forefront of the struggle to maintain the best possible medical care for the hospital's patients. Every member of the staff was cognizant of her devotion. She was repeatedly offered a post of honor as one of the officers of the Medical Board but always she declined, feeling that she could accomplish more though the obscurity of anonymity.

Dr. Stewart was also devoutly dedicated to the advancement of medical education for

* Prepared and published at the request of the Council of the College of Physicians of Philadelphia.

women. She served for many years as a member of the admissions committee of the Woman's Medical College, encouraged and helped many of its graduates to obtain desired internships and residencies, and later she would do everything in her power to help these young women physicians get started on a successful career. She was proud of her proteges' success. It was a great satisfaction to her that the plan to merge the Woman's Medical College with the Jefferson Medical School did not succeed.

Although she suffered considerably in the last few years of her life, her spirit, interest, and enthusiasm did not wane. In spite of considerable pain she attended committee meetings and continued her practice as long as her fast waning strength permitted. Never did she complain. Although she knew the nature of her illness and its incurability, she cheerfully chose to disregard the severity of her ill health and smilingly replied, when asked how she was, that she was bothered a little by some pains in the back which were probably arthritic but otherwise she felt fine.

Winifred Bayard Stewart's courage, devotion, and firm sense of right were an inspiration to all who were privileged to know this devoted woman physician.

Transactions of the Sections

SECTION ON GENERAL MEDICINE

27 October 1958

Spontaneous Remission of Metastatic Renal Cell Carcinoma: Case Report. John D. Hallahan, M.D. (Discusser: Thomas M. Birdsall, M.D.) (*Abstract* 1, below)

A Syndrome Associated with Alpha-Three Globulinemia. F. William Sunderman, Jr.*, M.D., and F. William Sunderman, M.D., Ph.D (Discusser: Harold L. Goldburgh, M.D.) (*Abstract* 2, below)

The Bromsulfalein Test—Its Uses and Abuses. Donald Berkowitz*, M.D. (Discusser: Henry J. Tumen, M.D.) (*Abstract* 3, below)

The Changing Picture in Thromboangiitis Obliterans. Meyer Naide, M.D., Michael M. Etzl, M.D., and Jacob S. Weiner*, M.D. (Discusser: Nicholas Padis, M.D.) (*Abstract* 4, below)

24 November 1958

The Effects of Total Body Irradiation on the Production of Antibody in Man. Irwin L. Stoloff*, M.D., Farid Haurani*, M.D., Evelyn Repplinger*, B.S., and W. Paul Havens, Jr., M.D. (Discusser: T. N. Harris, M.D.) (*Abstract* 5, below)

Clinical and Hemodynamic Changes Following Closure of Ventricular Septal Defects. Lamberto Bentivoglio*, M.D., Vladir Maranhao*, M.D., Harry Goldberg, M.D., and George Raber*, B.S. (Discusser: Daniel F. Downing, M.D.) (*Abstract* 6, below)

The Surgical Correction of Aortic Insufficiency by Bicuspidization of the Valve. Charles P. Bailey*, M.D., Dryden P. Morse*, M.D., Henry T. Nichols*, M.D. and Jacob Zimmerman*, M.B., F.R.C.S. (Discusser: William A. Likoff, M.D.) (*Abstract* 7, below)

Detection of Intestinal Bleeding with Chromium-51. Bernard Shapiro, M.D. (Discusser: Henry J. Tumen, M.D.) (*Abstract* 8, below)

26 January 1959

Primary Aldosteronism. Report of a Case. Dick D. Harrell*, M.D., and Harold L. Israel, M.D. (Discusser: James E. Clark, M.D.) (*Abstract* 9, below)

Hypoparathyroidism: Case Report and Review. William K. Frankenburg*, M.D., and Fred B. Rogers, M.D. (Discusser: Charles R. Shuman, M.D.) (*Abstract* 10, below)

Disseminated Lupus Erythematosus and Hypertension Without Renal Insufficiency. Alfred M. Sellers, M.D., Alton I. Sutnick*, M.D., William A. Jeffers, M.D., and Charles C. Wolferth, M.D. (Discusser: Ralph A. Jessar, M.D.) (*Abstract* 11, below)

Thalassemia—Hereditary Spherocytosis: Heretofore Undescribed Hereditary Hemolytic Anemia. Arthur Faulk*, M.D., and Abraham M. Frumin*, M.D. (Discusser: Harold A. Wurzel, M.D.) (*Abstract* 12, below)

SECTION ON MEDICAL HISTORY

21 October 1958

Meeting sponsored by the Hahnemann Medical College—fifth in the series arranged in conjunction with the medical schools of Philadelphia.

Homeopathy in Retrospect. Charles S. Cameron, M.D., Dean, Hahnemann Medical College.

Philadelphia Doctors in the White House. Dean Arvan*, A.B.

Second Annual Edward Bell Krumbhaar Award in Medical History. Presented to Donald E. Willard, Jr., M.D., for his essay: "W. W. Keen's "First Modern Brain Case".[1]

* By invitation.

[1] Published in *Transactions & Studies*, February, 1959.

17 February 1959

A Mock-Epic Poem, "La Guerre des Vénètes", by R. T. H. Laennec. William P. Boger*, M.D.

The Story of Herbs in Medicine. Douglas Macfarlan, M.D.

Dr. Robley Dunglison and Jefferson. Samuel X Radbill, M.D.

Mark Akenside: Eighteenth Century Physician-Poet. Herbert J. Dietrich, Jr., M.D.

SECTION ON OPHTHALMOLOGY[2]

23 October 1958

Joint Meeting of Section on Ophthalmology, College of Physicians of Philadelphia, and Eye Section, Philadelphia County Medical Society.

Cataract Extraction by the Modified Smith Technique. Richard A. Ellis, M.D.

Memoir of William Zentmayer (1864–1958). Warren S. Reese, M.D.

20 November 1958

Causes of Opacification of Corneal Grafts. A. Edward Maumenee*, M.D., The Johns Hopkins University. (Twenty-first Annual de Schweinitz Lecture)

11 December 1958

Spontaneous Recovery in a Case of Superior Oblique Tendon Sheath Syndrome of Brown. Francis Heed Adler, M.D.

Studies on Antibody Production in Rabbit's Eye. Joseph W. Hallett, M.D., and Irving H. Leopold, M.D.

Lens System Developed for Water to Air Vision in the Submerged Eye. T. D. Duane*, M.D.

15 January 1959

A Case of 'Total' Choroidal Detachment Following Cataract Surgery. H. Walter Forster, Jr., M.D.

Random Occurrence of Herpetic Disciform Keratitis in Rabbits. Theodore W. Sery*, Ph.D.

Repair of Orbital Deformity with Plastic Sponge Implant. Robert D. Mulberger*, M.D., and G. M. Shannon*, M.D.

19 February 1959

Surgical Treatment of Blepharoptosis. Raymond N. Berke*, M.D., Presbyterian Hospital, New York City.

SECTION ON OTOLARYNGOLOGY[3]

19 November 1958

The Clinical Significance of the Arrangement of the Sinuses in Sinusitis. Fred W. Dixon*, M.D., Cleveland, Ohio. (Discussers: Oscar V. Batson, M.D., and Austin J. Smith, M.D.) Joint Meeting of the Section on Otolaryngology, College of Physicians of Philadelphia, and The Philadelphia Laryngological Society.

21 January 1959

Repair of Facial Nerve Injuries. F. Boyes Korkis*, F.R.C.S., London, England. Joint Meeting of Section on Otolaryngology, College of Physicians of Philadelphia, and The Philadelphia Laryngological Society.

SECTION ON PUBLIC HEALTH, PREVENTIVE AND INDUSTRIAL MEDICINE

15 October 1958

Technical Cooperation—A Challenge![4] Eugene P. Campbell*, M.D. (Arthur Parker Hitchens Lecture III)

Panel Discussion on 'Poison Control Programs'. Joseph W. Spelman*, M.D. (Moderator), Howard M. Cann*, M.D., Harold Jacobziner*, M.D., Frederic Rieders*, Ph.D., Emil A. Tiboni*, M.P.H., and Felix E. Karpinsky*, M.D.

[2] Abstracts of the presentations at this Section customarily appear in the *American Journal of Ophthalmology* and *A.M.A Archives of Ophthalmology*.

[3] Abstracts of the presentations at this Section customarily appear in the *A.M.A. Archives of Otolaryngology*.

[4] Published in *Transactions & Studies*, February, 1959.

Joint Meeting, Section on Public Health, College of Physicians of Philadelphia, and Pennsylvania Public Health Association, Region One.

17 February 1959

HEALTH PROBLEMS AND SOCIAL DELINQUENCY. Hon. Richardson Dilworth, Mayor of Philadelphia, William A. Steiger, M.D., Maurice E. Linden*, M.D., Rev. W. Carter Merbreier*, and Randolph E. Wise, Esq.*

ABSTRACTS

Abstract 1

SPONTANEOUS REMISSION OF METASTATIC RENAL CELL CARCINOMA: CASE REPORT. John D. Hallahan, M.D.

The case of a 75-year old man having adenocarcinoma of the left kidney with local and pulmonary metastasis was presented. The pulmonary metastatic lesions spontaneously regressed after nephrectomy and remained absent for 33 months follow-up. The patient remained comfortable and was able to perform his daily work in the investment business without subsequent disability. He died in congestive heart failure from arteriosclerotic heart disease 33 months later. A complete pathological examination of the brain and body revealed no evidence of the previous tumor.

Postulations were advanced as to the cause of the regression of the metastatic lesions after the removal of the original tumor. No conclusive reason could be given as to why the metastatic lesions cleared following the resection of the original carcinoma.

This case illustrated the virtue of the removal of a parent renal tumor despite the evidence of extensive metastatic disease.

Abstract 2

A SYNDROME ASSOCIATED WITH ALPHA-THREE GLOBULINEMIA. F. William Sunderman, Jr., M.D., and F. William Sunderman, M.D., Ph.D.

Alpha-three globulin ("U" globulin) is a serum protein fraction with electrophoretic mobility between alpha-two and beta globulins. Alpha-three globulin has been demonstrated in normal sera by special technics for paper electrophoresis; with conventional procedures it is occasionally observed in sera from children and from patients with multiple myeloma, hepatic, or renal disease.

The authors have studied seven adult patients in whom persistent alpha-three globulinemia was associated with a chronic symptom complex suggestive of a collagen disease. These patients demonstrated myalgia, polyarthritis, dermatitis of the lower extremities, pleuritic pain, fever, emaciation and anemia. Other laboratory examinations, including L-E preparations and skin and muscle biopsies were normal. Three of the patients responded to therapy with cortisone. The possible relation of this syndrome to the collagen diseases was considered.

Abstract 3

THE BROMSULFALEIN TEST—ITS USES AND ABUSES. Donald Berkowitz, M.D.

Since the year 1925, the measurement of bromsulfalein retention has gained wide acceptance as a measure of hepatic function. This test is extremely sensitive and is often abnormal when all other liver function studies are normal. Its sensitivity, however, may actually be a disadvantage if the determination is not done correctly or is carried out in the presence of certain conditions which may themselves cause retention. Thus, improper technique in administration, failure to take blood at precisely the correct time interval following injection of the dye, previous feedings, and ambulation during the test period may influence the results. Furthermore, jaundice, fever, congestive failure, hemorrhage, shock, and recent administration of other dyestuffs are frequently responsible for misleading values.

In an effort to see how often in the average hospital the bromsulfalein test is performed in the presence of any of these circumstances, a review of 100 consecutive determinations was made at the Einstein Medical Center, Northern Division, Philadelphia, Pa.

It was found that in 73 cases, the test was carried out under improper conditions, and in 62 of these, an abnormal retention was found, in which instances the results must be viewed with some question.

It must be emphasized that the value of the bromsulfalein test as a screening procedure for liver disease is lost if the precise criteria for its performance are not adhered to.

Abstract 4

THE CHANGING PICTURE IN THROMBOANGIITIS OBLITERANS. Meyer Naide, M.D., Michael M. Etzl, M.D. and Jacob S. Weiner, M.D.

In recent years we have been making the diagnosis of thromboangiitis obliterans less and less often. The question arises whether this disease is occurring less frequently or whether we are eliminating conditions that we now know are not thromboangiitis obliterans. A number of case histories were presented in whom a diagnosis of thromboangiitis obliterans was made. These cases, we know today, are arteriosclerosis obliterans or embolism either from thrombi on plaques on the abdominal aorta or from the heart.

Our present concept of thromboangiitis obliterans is that it is a relapsing inflammation of arteries and veins occurring in men between the ages of 20 and 50 years, and occasionally also in women. The condition is probably a vascular sensitivity to various agents including tobacco, fungi that cause dermatophytosis, bacteria, or viruses that cause upper respiratory infections, and trauma.

The criteria for making the diagnosis of thromboangiitis obliterans include evidence of arterial disease in one or more extremities in a man between the ages of 20 and 50, and evidence for or a history of migrating phlebitis, and the smoking of at least 20 cigarettes a day. Involvement of arteries in the upper extremities is common in thromboangiitis obliterans and uncommon in arteriosclerosis obliterans. The migrating phlebitis of thromboangiitis obliterans must be differentiated from that seen in malignancy, gout and polycythemia. Arterial occlusion of thromboangiitis obliterans must be differentiated chiefly from arteriosclerosis obliterans and embolism. One must insert a word of caution, however, in that a young man with arterial disease or migrating phlebitis must stop smoking unless the diagnosis is proved to be not thromboangiitis obliterans but arteriosclerosis, gout, malignancy, or polycythemia. The views regarding this disease have gradually changed lately so that today it is diagnosed with much less frequency and must be considered a rare disease.

Abstract 5

THE EFFECTS OF TOTAL BODY IRRADIATION ON THE PRODUCTION OF ANTIBODY IN MAN. Irwin L. Stoloff, M.D., Farid Haurani, M.D., Evelyn Repplinger, B.S., and W. Paul Havens, Jr., M.D.

Numerous studies of the effects of roentgen radiation of the whole body on the immunologic response of animals have been made, and a considerable amount of precise information is available. In contrast, little is known about the effect of roentgen radiation on the immunologic response of man.

During recent attempts by certain members of this group and others to treat human leukemia with roentgen radiation of the whole body followed by transplantation of homologous marrow, an opportunity was presented to determine (a) whether the patients receiving varying dosages of radiation could produce antibody to one antigen (diphtheria toxoid), and (b) whether the transplanted homologous marrow cells could produce antibody to another antigen (tetanus toxoid) in their new environment. The anamnestic response was tested in both systems.

Six patients with acute leukemia and one patient with disseminated neuroblastoma were given roentgen radiation in amounts ranging from 170 r. to 800 r., calculated at the midplane of the body. Immediately following radiation, these patients received infusions of homologous marrow (5 to 20 billion cells) from donors who had shortly before been given a "booster" dose of tetanus toxoid. Four to

six days after radiation, the patients (all Schick-negative) were given a "booster" dose of diphtheria toxoid. The amounts of circulating diphtheria and tetanus antitoxins were measured at weekly intervals thereafter in the recipients, and the amounts of tetanus antitoxin were measured in the donors during the same period of study.

In five patients who received 170 to 490 r., the immunologic response was vigorous, and the maximum measured amounts of diphtheria antitoxin produced ranged from 18 to 150 units/ml. serum. Qualitatively, the pattern of response was similar to that found in normal persons, with a significant increase in antibody by the seventh day after inoculation of diphtheria toxoid, reaching a maximum in fourteen to twenty-eight days. The two patients who received 800 r. had no increase in their circulating diphtheria antitoxin, although in one patient this was measured only on the sixth day (time of death) after the injection of toxoid, and in the other on the sixth day and on the fourteenth day (time of death) after the injection of toxoid. The effects of radiation on the formed elements of the peripheral blood were qualitatively similar to those observed by others. There was no apparent relationship between the immunologic response and the degree of lymphopenia or neutropenia that occurred, and large amounts of antitoxin were produced during periods when circulating lymphocytes and neutrocytes were reduced to less than 50 cells/mm.^{3} of blood. These results indicate that the secondary immunologic response in man is not significantly depressed either qualitatively or quantitatively by total body radiation with as much as 490 r. at the midplane of the body. The fact that one subject who received 800 r. made no antibody fourteen days after a "booster" dose of diphtheria toxoid suggests, but does not prove, that this amount of radiation may be suppressive to secondary immunologic response under the conditions of this study.

The attempts to demonstrate the production of antibody (tetanus antitoxin) by the transplanted homologous cells in their new environment (irradiated patients) were unsuccessful. Six of these patients received cells from donors who produced tetanus antitoxin in amounts ranging from 12.5 units/ml. to more than 100 units/ml. serum. In spite of the fact that the donors were actively producing antibody at the time they furnished the transplants, none of the six recipients had any significant increase in the amounts of circulating tetanus antitoxin.

Abstract 6

Clinical and Hemodynamic Changes Following Closure of Ventricular Septal Defects. Lamberto Bentivoglio, M.D., Vladir Maranhao, M.D., Harry Goldberg, M.D., and George Raber, B.S.

Clinical, electrocardiographic, roentgenographic and hemodynamic data were obtained before and after closure of ventricular septal defect by open heart surgery in twelve patients; in five an associated pulmonary obstruction was removed.

The most common, but not constant, preoperative symptoms were fatigue and exertional dyspnea. They invariably disappeared after surgery.

Intermittent cyanosis disappeared postoperatively in the only patient with ventricular septal defect and pulmonic stenosis who displayed this sign. Harsh, grade II to IV systolic murmurs at the pulmonic and/or left precordial area disappeared post-operatively in all but one patient with ventricular septal defect, in whom it was markedly reduced and likewise greatly decreased in intensity in all patients with ventricular septal defect and pulmonic stenosis. Thrills, when present, constantly disappeared following surgery. The second pulmonic sound became normal or split in all cases.

The roentgenograms showed an inconstant increase of the heart size while electrocardiographic evidence of complete right bundle branch block invariably developed postoperatively, whatever the pre-operative pattern.

When pulmonary arterial and/or right ventricular hypertension and right ventricle-

pulmonary artery pressure gradients existed, they decreased or disappeared post-operatively. Systemic arterial oxygen saturation became normal in the only patient showing pre-operative evidence of unsaturation. The right to left and/or left to right shunts constantly disappeared and consequently the pulmonic blood flow was reduced post-operatively. The effective right ventricular work was, likewise, always reduced post-operatively while the pulmonary vascular resistance was generally unchanged.

From the above data it can be seen that repair of ventricular septal defects and correction of pulmonic stenosis, when present, constantly produced, in the present series, a significant reduction of the right ventricular pressure to normal or nearly normal values even when it represented 70% or more of the systemic arterial pressure.

In ventricular septal defect the fundamental factor responsible for the altered cardiac dynamics is the presence of an increased pulmonary blood flow. Pulmonary and right ventricular hypertension in this group was obviously not related to anatomic changes but rather to increased resistance due to vascular spasm.

The group of ventricular septal defect and pulmonic stenosis presented smaller left to right shunts and significantly higher right ventricular pressures. It is safe to assume that in these cases the obstruction at the pulmonic valve was the main cause of the right ventricular hypertension.

The disappearance of symptoms in the present series was clearly related to the post-operative reduction of pulmonic blood flow and pulmonary valvular resistance.

The development of complete right bundle branch block post-operatively was of no evident clinical consequence in the present series.

In conclusion, elevation of the pulmonary arterial and/or right ventricular pressures do not necessarily reflect organic pulmonary vascular changes and selection of patients for surgery cannot be determined on this basis alone. Open heart surgery, when indicated, is the treatment of choice for ventricular septal defects alone or associated with pulmonic stenosis.

Abstract 7

THE SURGICAL CORRECTION OF AORTIC INSUFFICIENCY BY BICUSPIDIZATION OF THE VALVE.

Charles P. Bailey, M.D.; Dryden P. Morse, M.D.; Henry T. Nichols, M.D.; and Jacob Zimmerman, M.B., F.R.C.S.

In attempting to devise a surgical solution for aortic regurgitation, we discovered that there were three serious problems to be overcome.

First, our understanding of the anatomy, physiology, and pathology of the aortic valve was seriously deficient.

Second, since closed surgical techniques early proved to be inadequate, it was necessary for the method of open heart surgery with an extracorporeal circulation to be developed before effective surgical techniques for correction of the leaky valve could be devised. Once this method of circulatory bypass was perfected, it became apparent that it also was necessary, during the period of definitive surgery, to maintain the vigor and tone of the heart muscle if recovery was to be assured. This can be accomplished best by individual cannulation of the coronary arteries so that they can be perfused continuously with arterialized blood by means of an additional pump.

Third, a method of effective repair of the damaged valve was essential if the patient was to be improved. Normally, each of the three deep, cup-like, delicate valve cusps fill with blood during closure. This distension causes them to protrude into the arterial stream in such a way that they come into precise mutual contact in a complex triradiate line, completely blocking the backward flow of blood. During ventricular ejection, the cusps empty and become flattened against the wall of the aorta, thus providing a passageway of maximal size. During closure the free margin of each of these cusps supports and is supported by those of its fellows. Should any one of these cusps become distorted or shrunken, not only

will it fail to defend its portion of the valve passageway, permitting regurgitation to occur, but the other two cusps, in the absence of its support, may "spill" and contribute to further leakage. Thus, rather moderate damage to even one cusp will throw a "monkey wrench" into the entire "precision mechanism" of the valve action.

After many trials and many failures, both in the animal laboratory and in tentative efforts to repair damaged human hearts in the autopsy room, it finally was realized that it is practically impossible to restore the "precision mechanism" of the normal tricuspid (aortic) valve action. However, the benign course observed in patients with congenital bicuspid aortic valves and our clinical experience in operating upon patients with acquired aortic stenosis (in many of whom the valve has become converted into a bicuspid structure), led to the thought that it might be possible to create a simpler bicuspid valve structure out of the irreparably destroyed tricuspid one. Such a valve would present but a single line of closure and would tend to close, in check-valve fashion, with the slightest backward surge of the blood. Naturally, such a valve would offer a somewhat greater amount of impedance to forward flow, but this would seem to be a small price to pay for restored competence.

In practice, this theory has proved eminently sound. However, since there are several types of valve pathology which can cause regurgitation, it was necessary to devise several different techniques for conversion of the damaged tricuspid structure into a bicuspid one (bicuspidization). These include excision of a third of the circumference of the aortic wall along with the non-coronary cusp, suture approximation of two adjacent cusps in order to convert them into a single conjoint structure, and the addition of pedicled strips of the aortic wall to the free margins of rigid and retracted cusps. Each case must be individualized in order to achieve maximal correction of the leak with the least possible narrowing of the passageway.

Abstract 8

Detection of Intestinal Bleeding with Chromium-51. Bernard Shapiro, M.D.

Measurements of intestinal blood loss have been made in a series of patients by measuring the amount of radioactivity appearing in the stools after the patient's circulating erythrocytes were labeled with radioactive chromium. The test appears to be a practical diagnostic procedure. Most of the patients were studied because of anemia of unknown origin and none had gross hemorrhage.

Where the Cr-51 technique demonstrated bleeding, it was episodic in nature and might have been missed if the stool collections had only been made for the usual 3 consecutive days. In several of the detected bleeders, the site of hemorrhage could not be found.

Abstract 9

Primary Aldosteronism. Report of a Case. Dick D. Harrell, M.D., and Harold L. Israel, M.D.

An instance of aldosteronism with some unusual features is reported.

A 48 year old nun was seen in March, 1958, at the Sacred Heart Hospital in Norristown, Pa., to which she had been admitted two weeks earlier. Her complaints were those of weakness, daily headaches, palpitation, and exertional dyspnea. There was no family history of hypertension, and she was known to be normotensive eight years prior to her hospital admission.

She had noticed unusual fatigue for six years, and during the preceding 18 months had complained of daily frontal headaches, exertional dyspnea, palpitation, urinary frequency, orthopnea, and very slight dependent edema. Hypertension had been discovered only 14 months prior to her hospital admission. Various drugs failed to control her hypertension or her symptoms.

The physical manifestations were those of sustained, severe hypertension of 230/140, slight cardiac enlargement, moderate accentuation of the aortic second sound, but no edema or signs of cardiac decompensation. Fundo-

scopic examination showed grade II retinopathy.

Investigation was carried out in detail, revealing the following studies to be normal: C.B.C., F.B.S., serology, T.P. and A./G., serum cholesterol, regitine tests, I.V. urogram, perinephric insufflation with air, cystoscopy, and urine cultures. The electrocardiogram showed early left ventricular hypertrophy and the chest x-ray only slight cardiac enlargement. All urinalyses showed an acid reaction, isosthenuria, and albuminuria. Electrolyte studies on numerous occasions revealed hypernatremia, hypokalemia and alkalosis; urinary excretion of sodium was low and potassium high. The 24 hour urinary excretion of aldosterone as determined by bioassay was slightly elevated.

The patient was further studied at the Clinical Center of the National Institutes of Health at Bethesda, Maryland. Increased plasma and urinary aldosterone were found but because of impaired renal function, there was considerable reluctance to attempt surgery. Exploration was finally performed in July, 1958, and both adrenal glands were found to be abnormal. Subtotal right and complete left adrenalectomy were carried out. The pathologic findings were those of bilateral nodular hyperplasia.

Six months after operation, the patient is working part time and is free of symptoms. Taking cortisone and reserpine, her blood pressure is normal and serum electrolytes are within normal range. However, blood urea nitrogen levels of 30 to 35 persist.

The dramatic and sustained response to subtotal adrenalectomy is best explained on the basis of primary aldosteronism.

Abstract 10

HYPOPARATHYROIDISM: CASE REPORT AND REVIEW. William K. Frankenburg, M.D., and Fred B. Rogers, M.D.

A case of hypoparathyroidism occurring in a 28 year old Negro woman was reported because of unusual features in diagnosis and treatment. A review of the entity of hypoparathyroidism, its clinical varieties and possible underlying metabolic defects, was presented in the light of problems encountered in this patient.

CASE REPORT

L. M., a 28 year old Negress, was admitted to Temple University Hospital on April 21, 1958, complaining of cramps in her arms and legs of one week's duration. She was in good health until one week prior to admission when she noticed gradual stiffening in her limbs with coexistent crampy pain. Her wrists would flex, fingers extend, and arms and legs effect athetoid movements lasting for about one minute and recurring every five or ten minutes. The patient recalled one similar episode of this type which had occurred at nine years of age following measles. The stiffening phenomenon disappeared spontaneously and did not recur until the present episode. This patient was the youngest of four children. Her three siblings were all taller in height than the patient. No other member of the family had a history of similar cramping or convulsions.

Physical examination revealed a short, fairly well-proportioned Negro woman showing facial roundness and fullness of cheeks. Approximately every five minutes she went through the dramatic spasmodic motions described above. She showed mental retardation (I.Q. 73 by Wechsler-Bellevue Intelligence Scale). Secondary sex characteristics were normal. The skeletal muscles were generally tense and it was difficult to straighten her fingers when in carpal spasm. Neurological examination was negative except for a Class I (very positive) Chvostek sign and a promptly positive Trousseau sign.

Blood pressure was 110/75 mm. Hg, and radial pulse 96 per minute. Her eyes revealed moderate bilateral, lenticular opacities. The teeth showed pitting and hypoplasia of the enamel, with marked diastemas. Several molar teeth had not erupted. The heart, lungs, and abdomen were normal. Hair and nails showed no atrophic changes and there were no areas of subcutaneous calcification.

Laboratory Data: Routine blood count and

urinalysis were normal. Blood Wassermann test was non-reactive. Serum calcium was 5.8 mg./100 cc., phosphorus 7.0 mg./100 cc., and magnesium 1.9 mg./100 cc. (normal 1–3). Alkaline phosphatase, total serum proteins and fractions, sodium, potassium, chloride, carbon dioxide, blood pH and BUN were normal. Special studies included: urinary 17 ketosteroids 7.0 mg./24 hrs. (normal 6-14); basal metabolic rate +14. Electrocardiogram QT interval was 0.44 seconds (normal 0.4 or below). Gastric analysis detected hyperchlorhydria, grade I, with an extragastric type of curve. Roentgenograms revealed calcification in the choroid plexus of the left lateral cerebral ventricle. The teeth showed generalized recession of the gingival tissues involving both soft tissue and bone. The first bicuspid was missing and the second bicuspid showed marked hypoplasia of the enamel—dating the onset of the process to about 7 years of age. The lower incisors were angulated laterally and separated by large calcareous deposits.

Several tests were performed for response to parathyroid hormone, according to the technique of Ellsworth and Howard. On three occasions following the injection of 200 units of parathyroid hormone (Lilly), the patient did not show a rise in the hourly urine phosphate excretion of more than 0.5 fold. In fact, there was a fall after two injections. (Controls showed a rise of only 2:5, 3, and 4.5 times the original—as compared with a rise of 5–6 times the preinjection level.) While these tests were being performed, the patient's serum calcium dropped to 4.2 mg./100 cc., and her serum phosphorus rose to 6.8 mg./100 cc. At this point, tetanic symptoms became so severe that she was given repeated doses of calcium gluconate intravenously, bringing the serum calcium up to 7.2 mg./100 cc., and phosphorus to 7.0 mg./100 cc. The patient was subsequently given daily intramuscular injections of 200 units of parathyroid hormone, as recommended by MacGregor. After eight days on this regimen, her serum calcium was 4.5 mg./100 cc., and phosphorus 6.6 mg./100 cc. Since the patient failed to respond to this program, she was next given Vitamin D 200–400,000 units per day, Amphojel 120 cc./day, and calcium lactate 12–16 gm. per day. This course was later supplemented by Hytakerol, 1.25 mg./day by mouth. The patient improved on this regimen. On June 2, the patient underwent right cataract extraction without complication. She was discharged from the hospital on June 15, 1958. Serum calcium was then 9.2 mg./100 cc., and phosphorus 5.6 mg./100 cc. She has continued under our observation to date, working as an hospital aide.

Review

Hypoparathyroidism was first described in 1891 by Gley, who described the symptoms of parathyroidectomy. Later, Howell (1899) and Loeb (1901) established the relationship between the parathyroid glands and the serum calcium level. Since this early work, there have been numerous reports of hypoparathyroidism following accidental excision of parathyroid tissue during thyroidectomy. Hypoparathyroidism, whether idiopathic or postoperative in type, shows the same symptom complex and response to injected parathyroid hormone.

Pseudohypoparathyroidism shows a clinical picture similar to hypoparathyroidism, plus certain other characteristics such as: round face, short wide-set fingers, early epiphyseal closure of metacarpal and metatarsal bones resulting in a shortening of digits, subcutaneous foci of ectopic ossification, and lack of response to parenteral administration of parathyroid extract. All of the above characteristics, however, are not necessarily present in each case. Our patient met the diagnostic criteria for a diagnosis of pseudohypoparathyroidism as recently outlined by Snapper et al. (*Medicine 37:* 317–352, Dec. 1958).

Still another clinical picture has been described including the above unusual physique and ectopic ossification, but with normal levels of serum calcium and phosphorus and no intracranial calcification. This entity has been given the confusing name of "pseudo-pseudo-hypoparathyroidism."

One can differentiate "pseudo-pseudo-hypo-

parathyroidism" from pseudohypoparathyroidism by determining serum calcium and phosphorus levels. Differentiation of the idiopathic from the pseudo type may be hindered, however, by difficulty in assessing the effect of parenteral parathyroid extract. One of the methods used to distinguish between these two entities is the Ellsworth-Howard test. This test measures the hourly urine phosphate output which increases if the kidneys respond to parathyroid extract. This increase is due to a lack of reabsorption of phosphate ions by the renal tubules. In cases of idiopathic hypoparathyroidism there is a ten-fold increase in the phosphate excretion (in euparathyroid state, a 5–6 fold increase). In pseudohypoparathyroidism there is only a two-fold increase, or none at all. Among objections raised to use of the Ellsworth-Howard test is the fact that urinary excretion of phosphate varies with the sleeping and waking pattern, changes in posture, meals, and the administration of insulin. Crawford and associates demonstrated that the phosphate excretion not only varies with the amount of parathyroid hormone, but also with the amount of phosphate filtered through the glomeruli. He suggested the effect of parathyroid hormone on the kidneys could be ascertained by determining tubular reabsorption of phosphate. This theory was modified by Kyle who stated that the effect of parathyroid hormone was better measured in hypoparathyroid patients by means of phosphate clearance. Another objection raised is that the parathyroid hormone has two active principles—one which raises the serum calcium (calcium mobilizing factor), and another which mobilizes phosphorus (phosphorus excretion factor). Since commercial preparations of parathyroid hormone are standardized by measuring their effect on serum calcium of dogs, it is postulated that there may be a destruction of the phosphorus excretion factor in preparation, accounting for a frequent lack of response in normal individuals. Two proteins have also been identified in parathyroid extract by electrophoretic studies. L'Heureux and Davies have separated two hormones from the parathyroid gland: one elevates the serum calcium and the other increases urinary phosphorus excretion. This duality may explain cases of so-called 'incomplete pseudohypoparathyroidism.' Another objection to the Ellsworth-Howard test, one which may explain the decreasing lack of response to parathyroid hormone, is that antihormones are formed by the body after its injection. This phenomenon may become manifest in some patients after a few days' treatment, in others after a few weeks or as long as five years. A review of the literature concerning the various types of hypoparathyroidism emphasizes the mootness of many questions in this complex disorder.

Abstract 11

Disseminated Lupus Erythematosus and Hypertension Without Renal Insufficiency. Alfred M. Sellers, M.D., Alton I. Sutnick, M.D., William A. Jeffers, M.D., and Charles C. Wolferth, M.D.

In this report we wish to emphasize the importance of the diagnosis of disseminated lupus erythematosus in young patients with hypertension who have not reached a stage of renal insufficiency. Although the majority of patients with lupus erythematosus show marked elevations of blood pressure only in association with severe kidney damage, this is not always true.

Case 1: B. E. was found to have hypertension at the age of 18 in 1956. His blood pressure was 210/150 mm. Hg. He showed fever, edema and moderate albuminuria. His blood urea nitrogen was 15 mgm. per cent. The phenolsulfonphthalein excretion test and intravenous urogram were normal. Three LE preparations were negative. A striking clnicali remission followed the use of steroid therapy.

One year later he was hospitalized because of fever, arthralgia and palmar skin lesions. His blood pressure was then 200/140 mm. Hg. A soft apical systolic murmur was present. Hepatosplenomegaly was noted. Again, renal function studies were within normal limits. Cramping abdominal pain occurred and was not relieved by steroid therapy. Death came

nine days later from gastrointestinal hemorrhage.

Necropsy revealed verrucous endocarditis of the mitral valve and renal lesions diagnostic of disseminated lupus erythematosus. The immediate cause of death was a massive hemorrhage from a gastric ulcer, possibly secondary to steroid therapy.

Case 2: H. T., whose hypertension was discovered in 1949 at the age of 18, failed to respond to various antihypertensive agents administered over a period of four years. She did not receive hydralazine. She suffered a single episode of rheumatic fever as a child. In 1953 her blood pressure was 230/170 mm. Hg. She showed papilledema. An apical diastolic murmur was heard. Renal function was good save for moderately impaired phenolsulfonphthalein excretion. A sub-total adrenalectomy and Adson sympathectomy were performed in 1953. Subsequently she received adrenal replacement therapy consisting of 37.5 mgm. cortisone, 2 mgm. desoxycorticosterone, and 3 gms. of sodium chloride per day. Hospitalization was advised in 1956 because of flank pain and symptoms of rheumatoid arthritis. At this time she was found to have anemia, leukopenia, thrombocytopenia and two positive LE preparations. Renal function was unchanged. Two weeks later she developed a facial rash and fever, her BUN rose to 79 mgm. per cent, and she shortly expired.

Necropsy revealed verrucous endocarditis of the mitral valve. Splenic arteries were compatible with the diagnosis of lupus erythematosus. Although the kidneys showed some wire looping, they were considered to show primarily malignant nephrosclerosis.

Although a normal blood pressure is considered characteristic of lupus erythematosus in the absence of azotemia, our patients illustrate the occurrence of marked hypertension relatively early in the course of the disease and before skin manifestations, hepatosplenomegaly or pancytopenia appear. The literature concerning lupus erythematosus does not mention this possibility.

Libman and Sachs first described sterile verrucous endocarditis in patients with lupus erythematosus, most of whom presented apical systolic murmurs. More rarely a diastolic murmur may be heard, as in our second patient, as a manifestation of verrucous lesions of the mitral valve.

Both patients with severe hypertension proved to have the cardiac and vascular lesions of lupus erythematosus. The absence of advanced renal insufficiency in these patients with lupus erythematosus and severe hypertension should alert us to look for disseminated lupus among patients with "essential hypertension."

Abstract 12

THALASSEMIA—HEREDITARY SPHEROCYTOSIS: HERETOFORE UNDESCRIBED HEREDITARY HEMOLYTIC ANEMIA. Arthur T. Faulk, M.D., and Abraham M. Frumin, M.D.

Thalassemia is an hereditary hemolytic anemia evidenced by the findings of hypochromia, microcytosis, orthochromatophilic target cells, ovalocytes and basophilic stippling (Wintrobe, M.: *Clinical Hematology*, ed. 4, pp. 654–664, 1956). Concomitantly present are increased resistance to hypotonic saline and variable amounts of A_2 hemoglobin (Josephson, A. M., et al.: *Blood 13:* 543–551, 1958). Thalassemia has been described in combination with several abnormal hemoglobins, i.e., C, E, H, and S (Singer, K.: *Am. J. Med. 18:* 633–652, 1955).

We have recently observed a family in which the grandfather (P. V.) has the findings of Thalassemia "trait"; his daughter (J. G.) has a blood picture which appears to be consistent with a combined disorder of Thalassemia "trait"—Hereditary Spherocytosis; and his grandson (S. G., Jr.) (the here-mentioned daughter's son) has the typical findings of Hereditary Spherocytosis. The father of the child has a normal blood picture. The child's two siblings (R. G. and D. G.) present findings which are difficult to classify as a disease process at present.

List of College Lectures, 1958

8 January 1958

Essex, Hiram E. Vasospasm— Its Important Bearing on Certain Problems of Circulatory Dynamics. (Nathan Lewis Hatfield Lecture XLII; *Transactions & Studies*, June, 1958)

5 February 1958

Moore, Robert A. Problems and Trends in Medical Education. (Mary Scott Newbold Lecture LXXVI; *Transactions & Studies*, June, 1958)

5 March 1958

Swineford, Oscar, Jr. Immunology of Clinical Allergy. (Hira S. Chouké Lecture II; *Transactions & Studies*, February, 1959)

2 April 1958

Burney, Leroy E. Health and Hospitals for an Aging Population. (James M. Anders Lecture XXXVII; *Transactions & Studies*, August, 1958)

14 May 1958

Joint Meeting of Philadelphia County Medical Society and College of Physicians of Philadelphia.

I. Presentation of the 35th Annual Strittmatter Award of the Philadelphia County Medical Society to Dr. Lewis C. Scheffey by Dr. I. S. Ravdin, Chairman of the *Award Committee*.

II. Presentation of citations to physicians who have completed fifty years in the practice of medicine. Citations awarded by Dr. John W. Shirer, President, Medical Society of the Commonwealth of Pennsylvania.

1 October 1958

Herbut, Peter A. Studies on the Properdin System in Transplantable Cancer. (Thomas Dent Mütter Lecture LXXI; *Transactions & Studies*, February, 1959)

5 November 1958

Thomas, Lewis, The Schwartzman Phenomenon: An Excerise in Experimental Pathology. (Mary Scott Newbold Lecture LXXVII). Joint Meeting, Philadelphia County Medical Society and College of Physicians of Philadelphia.

3 December 1958

De Bakey, Michael E. Surgery of the Aorta and Major Peripheral Arteries: Experience with 2,000 Cases. (Alvarenga Prize Lecture XVIII)

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1948. Braun, William, 406 Cooper St., Camden, N. J.
1953. Brav, Solomon S., 5575 N. Park Ave. (41)
1953. Breckenridge, Robert L., Our Lady of Lourdes Hosp., 1600 Haddon Ave., Camden 3, N. J.
1946. Briscoe, Clarence Conway, 811 Spruce St. (7)
1953. Brobeck, John R., Univ. of Pa. School of Medicine (4)
1958. Brody, Henry, Einstein Medical Center, York & Tabor Rds. (41)
1952. Brody, Morris W., 1930 Chestnut St. (3)
1948. Brogan, Edmund J., Thomas Wynne Apartment, Wynnewood, Pa.
1953. Brooke, John A., 1431 Spruce St. (2)
1958. Brooks, Frank P., Hospital of the Univ. of Pa. (4)
1936. Brown, Clark E., Lancaster Ave. & City Line Ave. (31)

1932. Brown, Claude P., 1930 Chestnut St. (3)
1959. Browneller, Ellsworth R., Jefferson Hospital (7)
1939. Brust, Raymond W., 4710 Chester Ave. (43)
1953. Bucher, Robert M., 3401 N. Broad St. (40)
1952. Bumgardner, Heath D., 3701 N. Broad St. (40)
1940. Burnett, W. Emory, 3401 N. Broad St. (40)
1954. Burros, Harry M., 255 S. 17th St. (3)
1941. Butler, Miriam, Watersmeet, Glen Mills, Pa.
1957. Calhoun, John Alfred, 214 Elm Ave., Swarthmore, Pa.
1958. Cameron, Charles S., 235 N. 15th St. (2)
1928. Campbell, Edward H., 133 S. 36st St. (4)
1952. Campbell, Edward W., 1318 Medical Arts Bldg. (2)
1944. Campion, Richard J., Penllyn Rd., Springhouse, Ambler, Pa.
1931. Cantarow, Abraham, 1025 Walnut St. (7)
1952. Cardone, John B., 831 N. 63rd St. (31)
1935. Carey, Lawrence S., 330 S. 9th St. (7)
1931. Cariss, Walter L., 51 N. 39th St. (4)
1956. Carnwath, John Wallace, Summit & Highland Aves., Jenkintown, Pa.
1930. Carpenter, Chapin, 1930 Chestnut St. (3)
1917. Carson, John B., Newtown Square, Pa.
1956. Carty, James Byron, 51 Hampden Rd., Upper Darby, Pa.
1956. Casey, Paul R., 8350 Roosevelt Blvd. (15)
1937. Castallo, Mario A., 1621 Spruce St. (3)
1943. Castigliano, S. Gordon, 853 Mt. Pleasant Rd., Bryn Mawr, Pa.
1949. Caswell, H. Taylor, 3401 N. Broad St. (40)
1954. Catenacci, Alfred J., 1417 S. Broad St. (47)
1954. Cathcart, Richard T., 739 S. Broad St. (47)
1957. Cattie, Vincent J., 4116 N. Broad St. (40)
1950. Chamberlain, Richard H., 3400 Spruce St.
1939. Chamberlin, George W., Reading Hospital, Reading, Pa.
1900. Chance, Burton, 9 Llanalew Road, Haverford, Pa.
1945. Chance, Burton, Jr., 8236 Germantown Ave. (18)
1958. Channick, Bertram J., 3521 N. Broad St. (40)
1954. Charny, Charles W., 2039 Delancey Pl. (3)
1947. Chouké, Kehar S., P. O. Box 447, Kecoughton, Va.
1945. Cianfrani, Theodore, 1930 Chestnut St.
1939. Ciccone, Emmet F., 2021 Murray St.
1951. Clagett, Augustus H. Jr., 1618 N. Broom St., Wilmington 6, Del.
1929. Clark, Eliot R., 315 S. 41st St.
1934. Clark, Jefferson Hamer, 101 Maple Ave., Wyncote, Pa.
1951. Clark, John K., 36th & Spruce Sts. (4)
1947. Clark, Thomas Williams, 9501 Germantown Ave. (18)
1947. Classen, Charles H., Cushman Rd. and Lancaster Ave., Rosemont, Pa.
1958. Closson, Edward W., 2320 S. Broad St. (45)
1910. Cloud, J. Howard, 111 St. Pauls Road, Ardmore, Pa.
1903. Coates, George Morrison, 1721 Pine St.
1959. Coghlan, William P., 1012 Walnut St. (7)
1957. Cohen, Frank L., 2113 Spruce St. (3)
1941. Cohen, J. Gershon, 255 S. 17th St.
1951. Cohen, Leon Solis-, 1530 Locust St. (2)
1949. Cohen, Louis B., 1320 Arrott St.
1907. Cohen, Myer Solis-, 2110 Spruce St.
1953. Cohen, Robert V., 520 Spring Ave., Elkins Park 17, Pa.

1955. Colcher, Abraham E., 269 S. 19th St. (3)
1934. Collins, Leon Howard, Jr., 133 S. 36th St.
1943. Colonna, Paul C., 133 S. 36th St.
1949. Conger, Kyril B., 3401 N. Broad St.
1945. Coonel, Pauline, 47 E. Mt. Pleasant Ave.
1930. Cooper, David A., 1520 Spruce St.
1953. Cooper, Donald R., 133 S. 36th St. (4)
1947. Coppolino, John F., 1807 S. Broad St.
1952. Corbit, John D., Jr., 3717 Chestnut St.
1959. Cormeny, George F., 3818 Chestnut St. (4)
1941. Cornelius, Chalmers E., 119 Summit Ave., Jenkintown, Pa.
1911. Cornell, Walter S., Board of Public Education, Parkway and 21st St.
1914. Corson, Edward F., 136 S. 16th St.
1946. Costello, Thomas J., 131 Pennsylvania Avenue, Bryn Mawr, Pa.
1924. Cowan, Alfred, 1930 Chestnut St.
1940. Cowan, Thomas H., 1930 Chestnut St.
1904. Craig, Frank A., 429 Montgomery Ave., Haverford, Pa.
1907. Crampton, George S., 2031 Locust St.
1947. Crane, A. Reynolds, 807 Spruce St.
1937. Crane, Martin P., 2412 N. Broad St.
1952. Crellin, J. Antrim, 1930 Chestnut St.
1955. Crist, Walter A., 211 N. 5th St., Camden, N. J.
1921. Crossan, Edward T., Buttonwood Hall, Conshohocken, Pa.
1937. Crump, Jean, 3300 Henry Ave. (29)
1959. Cullen, Milton L., 1321 W. Tabor Rd. (41)
1934. Custer, R. Philip, 126 Grays La., Haverford, Pa.
1958. D'Alonzo, Walter A., 1647 S. 17th St. (45)
1938. Dannenberg, Arthur M., 235 S. 15th St.
1951. Daugherty, Earl A., 2025 W. Girard Ave.
1953. Davie, John H., 269 S. 19th St. (3)
1947. Davis, C. Nelson, 133 South 36th St.
1936. Davis, David M., 255 S. 17th St.
1957. Davis, John Evan, Eastern Pa. Psychiatric Institute, Henry Ave. & Abbottsford Rd. (29)
1951. Davis, J. Wallace, 135 S. 18th St.
1937. Davis, Perk Lee, P. O. Box 701, Paoli, Pa.
1935. Deaver, J. Montgomery, Lankenau Medical Bldg. (31)
1957. Decker, John Paul, Pennsylvania Hospital (7)
1957. Dees-Porch, Frances, Glendale & Chestnut St., Upper Darby, Pa.
1947. Dehne, Theodore L., Adams Avenue and Roosevelt Blvd.
1935. Deibert, Irvin Elmer, 538 Cooper St., Camden, N. J.
1958. Deitz, George W. III, 1146 Arrott St. (24)
1923. De Long, Perce, 142 Princeton Rd., Bala-Cynwyd, Pa.
1955. De Long, Samuel L., 860 Lancaster Ave., Bryn Mawr, Pa.
1952. DeLuca, Charles Q., 255 S. 17th St.
1952. De Orsay, Ralph H., 1241 Lindale Ave., Drexel Hill, Pa.
1951. De Palma, Anthony F., 248 S. 21st St.
1959. DeTuerk, John J., 1930 Chestnut St. (3)
1928. Dewees, A. Lovett, Glen Mills, Del. Co., Pa.
1959. Dickens, Helen O., 1512 W. Oxford St. (21)
1958. Dickstein, Benjamin, 6810 Castor (24)
1956. Dietrich, Herbert James, Jr., 2035 Delancey Place (3)
1952. Digilio, Victor A., 2200 St. James Place.
1924. Dillon, Edward S., Conshohocken State Rd., Penn Valley, Narberth, Pa.

1953. Dixon, James P., Dept. of Public Health, City Hall Annex. (7)
1920. Doane, Joseph C., 617 W. Hortter St.
1948. Dohan, Francis Curtis, 80 Princeton Road, Cynwyd, Pa.
1954. Donaldson, James B., 235 N. Broad St. (2)
1946. Donnelly, Andrew J., 33rd and Powelton Ave.
1933. Donnelly, Joseph C., 6832 Ogontz Ave.
1937. Donnelly, Robert T. M., 255 S. 17th St.
1954. Downing, Daniel F., 8864 Norwood Ave. (18)
1952. Drayer, Calvin S., 111 N. 49th St.
1949. Dripps, Robert D., 3400 Spruce St.
1935. Duane, William, Jr., 2109 Spruce St. (3)
1956. Dubin, Isadore N., Woman's Medical College (29)
1944. Dublin, George J., 1930 Chestnut St.
1944. Dugger, John H., Episcopal Hospital (25)
1935. Duncan, Garfield G., 330 S. 9th St.
1937. Dunn, Charles W., Grange Ave., Collegeville, R. D. #1, Pa.
1951. Dunne, Francis S., 133 S. 36th St.
1953. Dupler, Donald A., 4028 Walnut St. (4)
1942. Durant, Thomas M., 3401 N. Broad St.
1947. Durkin, John Keenan, Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1942. Dyer, W. Wallace, Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1935. Eads, John T., 607 E. Durham St. (19)
1936. Easby, Mary Hoskins, Woman's Hospital.
1948. Eaton, Francis H., 4007 Chestnut St.
1937. Edeiken, Joseph, 2130 Delancey Pl.
1944. Eger, Sherman A., 2029 Delancey St.
1944. Ehrich, William E., 540 W. Hortter St.
1956. Eiman, John W., Abington Memorial Hospital, Abington, Pa.
1953. Eisenberg, Isadore J., 1520 N. 2d St., Harrisburg, Pa.
1959. Eisenhower, James S. D., Jr., 2704 Pacific Ave., Wildwood, N. J.
1958. Eisman, Sylvan H., 3600 Spruce St. (4)
1946. Elfman, Louis Kenneth, 6330 Lebanon Avenue.
1949. Elkinton, J. Russell, Oak Lane, Moylan, Pa.
1957. Ellis, Richard A., 269 S. 19th St. (3)
1955. Ellis, Samuel, 541 S. 46th St. (43)
1939. Ellis, Van Mashburn, 1528 Spruce St. (2)
1928. Ellison, Richard T., 8601 Stenton Ave.
1936. Ellson, J. Vernon, 807 Spruce St.
1904. Elmer, Walter G., 1801 Pine St.
1937. Elsom, Kendall A., Maloney Clinic Bldg., 36th & Spruce St. (4)
1934. Ely, William C., 3912 Chestnut St.
1932. Engel, Gilson C., Lankenau Medical Bldg. (31)
1945. English, O. Spurgeon, 3401 N. Broad St. (40)
1942. Erb, William H., 133 S. 36th St.
1957. Erdman II, William James, 3400 Spruce St. (4)
1950. Ersner, Matthew S., 1915 Spruce St.
1957. Etzel, Michael M., 9025 Frankford Ave. (14)
1954. Evans, William W., Box 2, Bryn Mawr, Pa.
1948. Everts, Glenn S., 5515 Wissahickon Ave., Apt. D-403.
1955. Eynon, Harold K., 538 Cooper St., Camden, N. J.
1946. Farell, David M., 1912 Spruce St.
1903. Farr, Clifford B., Railroad Ave. & County Line Rd., Bryn Mawr, Pa.
1945. Farrar, George Elbert, Jr., 528 Scott Rd., Gladwyne, Pa.

1953. Farrell, Harry L., 1930 Chestnut St. (3)
1930. Farrell, John T., Jr., 255 S. 17th St.
1935. Ferguson, Lewis Kraeer, 133 S. 36th St.
1936. Fetter, Ferdinand, 322 S. 21st St.
1945. Fetter, John S., 842 Worrell Rd., Jenkintown, Pa.
1935. Fetter, Theodore R., 255 S. 17th St.
1924. Fewell, Alexander G., 1924 Pine St.
1952. Fields, Harry, 133 S. 36th St.
1959. Fineberg, Charles, 255 S. 17th St. (3)
1955. Finestone, Albert J., 2039 Pine St. (3)
1943. Finkelstein, Arthur, 480 Latch's Lane, Merion, Pa.
1955. Finkelstein, David, 1930 Chestnut St. (3)
1955. Finley, John K., 527 Welsh St., Chester, Pa.
1954. Finn, Joseph L., 7109 Rising Sun Ave. (11)
1947. First, Arthur, 1714 Spruce St.
1954. Fischer, Carl C., 95 Overhill Rd., Cynwyd, Pa.
1958. Fisher, Robert M., 118 Airdale Rd., Rosemont, Pa.
1945. Fiske, Frederick A., 1530 Locust St.
1956. Fite, Franklin K., Germantown Hospital (44)
1952. Fitts, William T., Jr., 3400 Spruce St.
1928. Fitz-Hugh, Thomas, Jr., 2016 Delancey Pl.
1952. Flexner, Louis B., Univ. of Pa. School of Medicine (4)
1928. Flick, John B., 819 Black Rock Rd., Gladwyne, Pa.
1954. Flick, John B., Jr., Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1952. Flinn, Lewis B., 503 Delaware Ave., Wilmington, Del.
1939. Flippin, Harrison F., Lankenau Hospital (30)
1957. Forman, Simon B., 255 S. 17th St. (3)
1957. Fornwalt, George R., Glendale & Chestnut Sts., Upper Darby, Pa.
1954. Forster, H. Walter, Jr., 37 S. 20th St. (3)
1941. Fox, C. Calvin, 1806 Spruce St.
1906. Fraley, Frederick, 6005 Overbrook Ave. (31)
1957. Frankel, Donald S., 255 S. 17th St. (3)
1955. Frayer, William C., 313 S. 17th St. (3)
1941. Frazier, William Doane, 419 Gilpin Rd., Narberth, Pa.
1952. Freed, Herbert, 255 S. 17th St.
1956. Freedman, Joseph T., 8-A Rittenhouse Plaza (3)
1958. Freiwald, Milton J., 1530 Spruce St. (2)
1953. Fried, Paul H., 1812 Spruce St. (3)
1955. Friedenberg, Zachary B., 133 S. 36th St. (4)
1951. Friedman, Paul Sigmund, 1422 Chestnut St.
1958. Frisch, Frederick, 40 Morris Ave., Bryn Mawr, Pa.
1933. Fritch, James Scott, 269 S. 19th St.
1937. Fritz, Herbert H., Pennswood Rd., Bryn Mawr, Pa.
1953. Fruchter, Joseph M., 601 W. Cliveden St., C-221 (19)
1940. Fry, Kenneth E., 1719 Rittenhouse Sq.
1935. Fry, Wilfred Eyles, 1930 Chestnut St.
1937. Furlong, Thomas F., Jr., Times Medical Building, Ardmore, Pa.
1954. Gambescia, Joseph M., 255 S. 17th St. (3)
1935. Gammon, Geo. D., Univ. of Pa. Hospital (4)
1951. Garlichs, Richard W., 216 N. Manoa Road, Havertown, Pa.
1931. Garner, Vaughn C., Germantown Professional Building
1953. Garnet, James D., 807 Spruce St. (7)
1959. Gartland, John J., 269 S. 19th St. (3)

1950. Garvin, Eugene J., 533 S. 46th St. (43)
1953. Geckeler, Edwin O., 269 S. 19th St. (3)
1958. Geckeler, George D., 230 N. Broad St. (2)
1952. Gefter, William I., 366 Penn Rd., Wynnewood, Pa.
1935. Geist, Donald C., 1930 Chestnut St.
1955. Gelfand, David, 1722 Pine St. (3)
1954. German, George B., 429 Cooper St., Camden, N. J.
1955. Gettes, Bernard C., 128 Overhill Rd., Cynwyd, Pa.
1934. Gibbon, John H., Jr., 1025 Walnut St.
1939. Gibson, Glen Gregory, 2031 Locust St.
1944. Gifford, Edward S., Jr., 1913 Spruce St.
1954. Gilbert, Philip D., Cooper Hospital, Camden, N. J.
1954. Giletto, Basil J., 2020 Locust St. (3)
1920. Gill, A. Bruce, 1603 Normandy Drive, Mt. Dora, Fla.
1956. Gill, Robert J., 801 Spruce St. (7)
1927. Gilmour, William R., 6616 Woodland Ave. (42)
1952. Gilpin, Sherman F., Jr., 3701 N. Broad St.
1949. Ginsburg, Isadore W., 3401 N. Broad St. (40)
1931. Giordano, Anthony A. S., 129 Carpenter Lane (47)
1958. Girsh, Leonard S., 3701 N. Broad St. (40)
1954. Gislason, Gerhard J., Medical Arts Bldg. (2)
1954. Glover, Robert P., 269 S. 19th St. (3)
1956. Goldberg, Harry, 249 N. Broad St. (2)
1933. Goldburgh, Harold L., 1932 Spruce St.
1935. Goldsmith, N. Ralph, 1351 Tabor Rd.
1958. Golub, Leib J., Medical Arts Bldg. (2)
1949. Gopadze, Illarion I., 255 S. 17th St. (3)
1956. Goracci, Armando F., 2040 Poplar St. (30)
1937. Gordon, William, 5345 Spruce St.
1956. Gouley, Benjamin, 5577 N. Park Ave. (41)
1941. Gouterman, Joseph I., 1930 Chestnut St.
1957. Grahn, Henry Victor, 5132 Chester Ave. (43)
1926. Grant, Francis Clark, 750 Germantown Pike, Lafayette Hill, Pa.
1958. Greco, Toby A., 2506 S. 20th St. (45)
1940. Greene, Lloyd B., 803 Spruce St. (7)
1952. Greening, Roy R., 3400 Spruce St.
1958. Greenspan, Benjamin, 1319 W. Tabor Rd. (41)
1955. Greenstein, Raphael H., 1710 Pine St. (3)
1934. Griffith, John Q., Jr., 6 N. Fredericksburg Ave., Margate, N. J.
1945. Griffiths, John E., Mermont Apartments, Bryn Mawr, Pa.
1941. Groff, Robert A., Univ. of Pa. Hospital (4)
1952. Grotzinger, Paul J., 230 N. Broad St. (2)
1952. Grove, D. Dwight, 230 N. Broad St.
1945. György, Paul, Univ. of Pa. School of Medicine (4)
1935. Hadden, Samuel B., 250 S. 18th St. (3)
1951. Hafkenschiel, Joseph H., Jr., 302 Lankenau Medical Bldg. (31)
1945. Hahn, George A., 255 S. 17th St.
1935. Haines, Harlan F., 134 S. State Rd., Upper Darby, Pa.
1955. Hallahan, John D., 104 W. Front St., Media, Pa.
1951. Hallett, Joseph W., 1001 Medical Arts Bldg., 16 & Walnut Sts.
1939. Hamblock, Leonard C., 1632 Spruce St.
1954. Hand, B. Marvin, 269 S. 19th St. (3)
1938. Hand, John G., 1724 Pine St.

1947. Hanes, William James, 218 E. Beechtree Lane, Wayne, Pa.
1958. Hanlon John J., 519 City Hall Annex (7)
1950. Hansen, A. Victor, 3401 N. Broad St. (40)
1957. Harasym, Emil L., 952 N. Franklin St. (23)
1956. Harbert, Frederick, Jefferson Hospital (7)
1952. Harer, W. Benson, State Rd. & Rogers Ave., Bywood, Pa.
1951. Harkins, Herbert P., 1930 Chestnut St.
1959. Harris, James S. C., 5401 Wayne Ave. (44)
1932. Harris, Stanley E., 229 Maple Ave., Delanco, N. J.
1948. Harris, T. N., 5112 Woodbine Ave. (31)
1929. Harrison, Francis G., 1900 Spruce St.
1926. Hartmann, Fred L., 1914 Pine St.
1946. Harvey, Edith E., 406 York Rd., Jenkintown, Pa.
1947. Harvey, Rolfe Marsh, 669 Mill Rd., Villanova, Pa.
1935. Haskell, Benjamin F., 1427 Spruce St.
1946. Hatfield, C. Alexander, 330 S. 9th St.
1958. Haupt, George J., 1012 Walnut St. (7)
1947. Havens, Walter Paul, Jr., 300 Cherry Lane, Wynnewood, Pa.
1947. Hawthorne, Herbert Reid, 255 S. 17th St. (3)
1952. Hayllar, Benjamin L., 803 Spruce St. (7)
1957. Hedges, Thomas R., Jr., 1930 Chestnut St. (3)
1946. Hendrickson, Frank O., 140 N. Rolling Rd., Springfield, Pa.
1953. Henry, George C., 3400 N. Broad St. (40)
1944. Herbut, Peter A., Jefferson Medical College Hospital
1951. Herrman, Clinton S., 1301 Spruce St. (7)
1954. Herron, James R., 1486 Haddon Ave., Camden 3, N. J.
1957. Hess, Catherine B., 4 W. Mt. Pleasant Ave. (19)
1924. Hewson, William, 1614 Locust St.
1949. Heyl, W. Meredith, 100 W. Coulter St.
1956. Hildreth, Eugene A., University of Penna. Hospital (4)
1952. Hinkson, DeHaven, 329 N. 40th St.
1924. Hirst, John C., 2d., 500 N. 20th St.
1943. Hitschler, William J., Bethlehem Pike & Summit (18)
1944. Hneleski, Ignatius S., 802 S. 48th St.
1943. Hockaday, Agnes, 316 Shadeland Ave., Drexel Hill, Pa.
1939. Hodes, Philip J., Univ. of Pa. Hospital (4)
1947. Hodges, John H., 1025 Walnut St.
1948. Hoffman, George L., Jr., 133 S. 36th St.
1954. Hollander, George, 3500 Vista St. (36)
1949. Hollander, Joseph L., 36th & Spruce Sts. (4)
1932. Hollingsworth, I. Pemberton, 411 N. Walnut St., West Chester, Pa.
1951. Hollis, Charles B., 1930 Chestnut St.
1952. Holloway, Edward E., 1739 W. Montgomery Ave.
1949. Hopkins, Henry U., Garden Court Apts., 47th & Pine Sts.
1953. Horan, Charles A., 734 N. 63rd St. (31)
1946. Horan, Gerald W., 5819 Chester Ave.
1947. Horwitz, Orville, Hospital of University of Pa. (4)
1950. Houlihan, Carl T., 728 S. 60th St.
1944. Housel, Edmund L., 255 S. 17th St.
1928. Houser, Karl Musser, 2035 Delancey Pl.
1958. Howard, John M., 230 N. Broad St. (2)
1948. Howland, Alvin Wilcox, 255 S. 17th St.
1946. Howson, John Yeatts, 316 Lancaster Ave., Wayne, Pa.

1951. Hubbard, John P., University of Penna. School of Medicine.
1947. Huber, John Franklin, Temple University School of Medicine. (40)
1937. Hughes, Joseph, 111 N. 49th St.
1939. Hughes, P. Boland, 419 Hillbrook Rd., Bryn Mawr, Pa.
1951. Hundley, J. Warren, 3818 Chestnut St.
1954. Hunsicker, William C., Jr., 255 S. 17th St. (3)
1946. Hunt, William T., Jr., 6 Penn Center Plaza (3)
1921. Hunter, Robert John, 928 N. 63rd St. (31)
1952. Hyman, Harold L., 3401 N. Broad St. (40)
1950. Imbriglia, Joseph E., 329 Shadeland Ave., Drexel Hill, Pa.
1952. Imperiale, C. Charles, 1331 Pine St.
1949. Ingraham, Norman R., Jr., Stenton and Whitemarsh Ave.
1957. Isaacson, Howard, Haverford Ave. & Drexel Rd.
1951. Isard, Harold J., Mt. Sinai Hospital, 5th & Reed Sts.
1951. Israel, Harold L., 304 S. 19th St. (3)
1947. Israel, S. Leon, 2116 Spruce St.
1952. Iverson, Preston C., 1930 Chestnut St.
1932. Jackson, Chevalier L., 3401 N. Broad St.
1934. Jacobs, Maurice, 1831 Delancey Place.
1947. Jaeger, Rudolph, Jefferson Medical College Hospital.
1952. Janton, O. Henry, 2008 Walnut St. (3)
1941. Jeffers, William A., University of Pa. Hospital. (4)
1942. Johnson, Julian, 3400 Spruce St. (4)
1959. Johnson, Robert G., 1012 Walnut St. (7)
1938. Johnson, Thomas A., 135 S. 18th St. (3)
1952. Jonas, Karl C., 2601 Parkway.
1924. Jones, Harold W., 1930 Chestnut St.
1957. Jordan, Luke W., 1486 Haddon Ave., Camden 3, N. J.
1957. Kacher, Leon, 2037 Pine St. (3)
1936. Kain, Thomas M., 403 Cooper St., Camden, N. J.
1957. Kane, Sydney H., 3458 Cottman Ave. (49)
1948. Kaplan, Louis, 2040 Pine St.
1938. Kasper, Kelvin A., 135 S. 18th St.
1958. Kasser, Max D., 37 S. 20th St. (3)
1945. Katz, G. Henry, 111 N. 49th St.
1958. Katz, Jacob, Medical Arts Bldg. (2)
1941. Kauffman, M. Luther, Medical Arts Bldg., Jenkintown, Pa.
1947. Kay, Calvin Frederick, Univ. of Pa. Hospital. (4)
1954. Kaye, Robert, Children's Hospital, 1740 Bainbridge St. (46)
1947. Keefer, George Pfahler, 3708 Woodland Ave., Drexel Hill, Pa.
1949. Keiserman, Joseph, 5460 Baltimore Ave.
1935. Kelly, George F. J., N.E. Cor. 20th and Chestnut Sts.
1932. Kelly, Herbert T., 1900 Spruce St.
1941. Kennedy, Patrick J., 32 Hampden Rd., Upper Darby, Pa.
1947. Kennedy, William Blair, Univ. of Pa. School of Medicine. (4)
1948. Kern, Franklin Moore, 903 Nicholson Rd., Wynnewood. Pa.
1921. Kern, Richard A., Temple University Hospital. (40)
1935. Keyes, Baldwin L., 2031 Locust St.
1930. Kimbrough, Robert A., Jr., 807 Spruce St.
1936. King, Orville C., 330 S. 9th St.
1958. Kirby, Charles K., 3400 Spruce St. (4)
1952. Kirshner, Jacob J., 1930 Chestnut St.
1920. Klauder, Joseph V., 1934 Spruce St.

1920. Klein, Thomas, 250 S. 18th St.
1952. Kleinbart, Morris, 1922 Pine St.
1954. Kligman, Albert M., 36th & Spruce Sts. (4)
1934. Kline, Oram R., 514 Cooper St., Camden, N. J.
1959. Kline, Oram R., Jr., 524 Cooper St., Camden, N. J.
1948. Klingensmith, Paul O., 133 S. 36th St.
1958. Klingensmith, Walter C., 3600 Spruce St. (4)
1956. Klinghoffer, June F., 5501 Green St. (44)
1932. Klopp, John W., Gwynedd Valley, Pa.
1919. Knipe, Norman L., 514 Baird Rd., Merion, Pa.
1959. Knowles, Harry J., 2029 Delancey St. (3)
1945. Kobler, H. B., 658 N. 63rd St.
1914. Kolmer, John A., Cynwyd, Pa.
1957. Koltes, John A., Jefferson Hospital, 14th Annex, 10th & Sansom St. (7)
1936. Konzelmann, Frank Williamson, Shore Memorial Hosp., Somer's Point, N. J.
1949. Koop, C. Everett, 1740 Bainbridge St.
1952. Kornblueh, Igho H., Welsh Rd , & Verree Rd. (15)
1958. Kovach, Coleman W., 2031 Locust St. (3)
1941. Krall, J. Thomas, 1927 Spruce St.
1948. Kramer, David W., 2007 Pine St.
1954. Krasnoff, Sidney O., Tabor Medical Bldg. (41)
1944. Krauss, Frederick Harold, 16th & Walnut Sts.
1952. Kravitz, Charles H., 626 Ashbourne Rd., Elkins Park, Pa.
1949. Kremer, David N., 1918 Pine St.
1949. Kressler, Robert J., 330 S. 9th St.
1941. Krewson, William E., 3rd., 1930 Chestnut St.
1914. Krumbhaar, Edward B., P. O. Box 4056 (18)
1947. Kuhlenbeck, Hartwig, Woman's Medical College.
1947. La Boccetta, Alfred Charles, Phila. General Hospital, 34th St. & Curie Ave. (4)
1952. Lachman, John W., 3401 N. Broad St. (40)
1949. La Clair, Charles H., Jr., 1021 W. Main St., Norristown, Pa.
1952. Lafferty, Henry D., 37 S. 20th St.
1951. Lame, Edwin L., 51 N. 39th St.
1950. Lamont, Austin, 3400 Spruce St. (4)
1946. Lampe, William T., 4207 Tyson Ave.
1952. Lang, Warren R., 1012 Walnut St. (7)
1941. Langner, Paul H., Jr., 1208 Edmonds Ave., Drexel Hill, Pa.
1945. Lansbury, John, 3401 N. Broad St. (40)
1926. Laws, George M., 1907 Spruce St.
1936. Leaman, William G., Jr., Presidential Apts: Adams House B-909.
1950. Learner, Norman, 3701 N. Broad St. (40)
1920. Leavitt, Frederic Headley, 1527 Pine St.
1957. Lee, Charles Trumbull, Jr., 1 Summit St. (18)
1941. Leberman, Paul R., 136 S. 16th St.
1942. Lehman, James A., 255 S. 17th St.
1952. Lehman, J. Stauffer, 230 N. Broad St.
1951. Leivy, Frank E., 1321 Spruce St.
1944. Lell, William A., 333 S. 18th St. (3)
1935. Lemmon, William T., 133 S. 36th St.
1943. Leopold, Irving H., 1711 Rittenhouse Sq. (3)
1955. Leopold, Robert L., 269 S. 19th St. (3)
1944. Lettiere, Anthony J., 425 E. State St., Trenton, N. J.
1933. Levering, J. Walter, Abington Memorial Hospital, Abington, Pa.

1959. Levick, Leonard J., 1300 E. Cliveden St. (19)
1958. Levine, Samuel, 7100 N. 19th St. (38)
1958. LeWinn, Edward B., 1351 Devereaux Ave. (11)
1915. Lewis, Fielding O., Providence Road, Media, Pa.
1938. Lewis, James P. 3815 Chestnut St.
1955. Lieberman, George E., 2019 Pine St. (3)
1956. Likoff, William, 249 N. Broad St. (2)
1940. Limberger, William A., 301 S. Church St., West Chester, Pa.
1958. Lin, David Y. P., 2228 S. Broad St. (45)
1938. Lindauer, M. August, 133 S. 36th St.
1956. Lindquist, John N., 422 Sabine Ave., Wynnewood, Pa.
1927. Lindsey, Walter H., 325 Callowhill St., Perkasie, Pa.
1936. Lintgen, Charles, 1930 Chestnut St.
1958. Lipschutz, Arthur, 7974 Fayette St. (19)
1953. Lipsius, Edward I., 319 S. 16th St. (3)
1945. Lisker, Samuel, 255 S. 17th St. (3)
1959. Little, Ralph B., 111 N. 49th St. (39)
1944. Livezey, Mary M., 7200 Cresheim Rd.
1936. Llewellyn, Thomas H., 910 N. 63rd St.
1907. Lodholz, Edward, The Fairfax, 43rd & Locust Sts.
1958. Loftus, Thomas A., 1025 Walnut St. (7)
1951. Long, Joseph P., 1930 Chestnut St.
1935. Long, William L., 2038 Locust St. (3)
1931. Longaker, Edwin P., 41 E. Montgomery Ave., Ardmore, Pa.
1951. Lorber, Stanley H., 3701 N. Broad St. (40)
1936. Lucchesi, Pascal Francis, 601 E. Gorgas Lane.
1932. Lukens, Francis D. W., Univ. of Pa. Hospital (4)
1939. Luongo, Romeo A., 2054 Locust St.
1947. Lurie, Max B., Henry Phipps Institute, 7th and Lombard Sts.
1951. Luscombe, Herbert A., 136 S. 16th St.
1952. Lutman, Frank C., 100 W. Coulter St.
1950. Lynch, J. Edward, 23 E. Wynnewood Rd., Wynnewood, Pa.
1953. Lyons, John W., 255 S. 17th St. (3)
1952. McCabe, Edward S., 136 S. 16th St. (2)
1932. McCahey, James F., 222 S. 19th St. (3)
1936. McCarthy, Patrick A., 229 E. Price St. (44)
1958. McClenahan, John L., 39 E. Chestnut Hill Ave. (18)
1935. McClenahan, William U., 1 Summit Ave.
1953. McCloskey, Joseph F., Jefferson Hospital. (7)
1955. McClure, Carroll R., 17 S. Church St., W. Chester, Pa.
1957. McConnell, Edward L., Jr., 2230 S. Broad St. (45)
1934. McCouch, Grayson P., Univ. of Pa. School of Medicine (4)
1938. McCrea, Lowrain E., 1930 Chestnut St.
1958. McCune, Wallace G., Germantown Hospital (44)
1937. McCutcheon, Morton, Cambridge Apts.
1942. McDonald, P. Robb, 1930 Chestnut St.
1953. McDonnell, William V., 1025 Walnut St. (7)
1952. McElroy, Robert C., 133 S. 36th St.
1950. McFadden, William M., 1187 E. Washington Lane
1949. McGavic, John S., 601 Montgomery Ave., Bryn Mawr, Pa.
1945. McGee, Lemuel Clyde, Hercules Powder Company, Wilmington, Del.
1954. McGehee, Edward H., 15 E. Highland Ave. (18)
1952. McGlade, Thomas H., 719 Cooper St., Camden, N. J.

1956. McGrath, Raymond J., 1720 Spruce St. (3)
1959. McKeown, John J., 1012 Walnut St. (7)
1933. McLaughlin, James S., 330 S. 9th St.
1924. McMillan, Thomas M., 1901 Walnut St. (3)
1924. McPhedran, F. Maurice, Germantown Hosp., Penn and Chew Sts.
1955. McStravog, Lawrence J., 6910 Market St., Upper Darby, Pa.
1954. MacFadyen, Bruce V., 135 S. 18th St. (3)
1943. Macfarlan, Douglas, 1805 Chestnut St.
1932. Macfarlane, Catharine, 136 S. 16th St.
1947. Machella, Thomas E., Univ. of Pa. Hospital. (4)
1950. Mackmull, Gulden, Greene & Coulter St. (44)
1949. MacNeal, Perry Scott, 330 S. 9th St.
1939. Macneill, Norman M., 5807 Chew St.
1951. Mac Neish, W. J., Medical Arts Bldg., 16th & Walnut Sts.
1955. Macy, Dorothy, Jr., 3 Beechwood Rd., Pine Ridge, Media, R. D. 3, Pa.
1957. Madow, Leo, 111 N. 49th St. (39)
1935. Maeder, LeRoy M. A., 1910 Rittenhouse Sq.
1952. Mahoney, J. Francis, 250 S. 18th St.
1942. Manges, Lewis C., Jr., 2001 Delancey Place (3)
1954. Manges, Willis E., Methodist Hospital, Broad & Wolf Sts. (48)
1936. Mann, Bernard, 2019 Pine St.
1956. Manstein, George, 1351 Tabor Rd. (41)
1953. Marbach, A. Herbert, 1307 Tabor Rd. (41)
1948. Marden, Philip Ayer, 1910 Spruce St. (3)
1937. Margolies, Alexander, 1351 Tabor Rd.
1958. Marino, Daniel J., 269 S. 19th St. (3)
1950. Mark, George E., 3401 N. Broad St. (40)
1950. Marshall, E. Wayne, Jr., 1520 Spruce St.
1951. Martin, William L., Hahnemann Hospital.
1952. Martucci, Albert A., 1460 E. Cheltenham Ave.
1953. Marx, Halvey E., 282 N. Lansdowne Ave., Lansdowne, Pa.
1953. Massey, Franklin C., 136 S. 16th St. (2)
1935. Matthews, Robert A., 1025 Walnut St. (7)
1939. May, Hans, 255 S. 17th St.
1951. Mayock, Robert L., 3600 Spruce St. (4)
1958. Mays, Ralph Whiteman, 154 W. Tulpehocken St. (44)
1952. Mecray, Paul, Jr., 405 Loucroft Ave., Haddonfield, N. J.
1951. Medinger, Frederick G., 255 S. 17th St.
1955. Melnick, Theodore, 1152 N. 63rd St. (31)
1941. Mendell, Theodore H., 2023 Spruce St.
1951. Mendelssohn, Edwin, 1301 Tabor Rd. (41)
1949. Menkin, Valy, 3401 N. Broad St.
1943. Meranze, David R., 1031 Lakeside Ave.
1956. Merves, Louis, 1930 Chestnut St. (3)
1955. Mervine, Thomas B., 133 S. 36th St. (4)
1959. Metzger, Harry N., 1815 Pine St. (3)
1955. Meyer, Eugene A., 1438 Mt. Ephraim Ave., Camden, N. J.
1937. Meyer, George Phillip, 410 Haddon Ave., Camden, N. J.
1956. Miller, Bernard Joseph, 340 S. 15th St. (3)
1935. Miller, Ford A., 4016 Chestnut St.
1939. Miller, Malcolm W., Lankenau Medical Bldg. (31)
1939. Miller, Merle M., 6013 Greene St.
1932. Miller, M. Valentine, 114 W. Phil-Ellena St.

1921. Miller, T. Grier, 133 S. 36th St.
1955. Miller, William H., Fitzgerald Mercy Hospital, Darby, Pa.
1952. Minehart, John R., 4821 Germantown Ave.
1952. Mintz, S. S., 1930 Chestnut St. (3)
1937. Missett, Joseph Vincent, Jr., 2031 Locust St.
1904. Mitchell, Charles Franklin, 2003 Pine St.
1950. Mitchell, John McK., Univ. of Pa. School of Medicine. (4)
1952. Mitchell, Robert McNair, 330 S. 9th St.
1932. Mohler, Roy W., 1806 Spruce St.
1936. Montgomery, Hugh, 119 Glenn Road, Ardmore, Pa.
1936. Montgomery, John B., 1930 Chestnut St.
1932. Montgomery, Thaddeus L., 2031 Locust St.
1935. Moore, John Royal, 3401 N. Broad St.
1947. Moore, Matthew Thibaud, 1813 Delancey Pl.
1958. Moore, Samuel R., Jr., 1600 Arch St. (1)
1958. Morani, Alma D., 2665 Midvale Ave. (29)
1936. Morgan, David R., Jefferson Medical College.
1944. Morris, Harold H., Villa Nova, Pa.
1945. Morris, I. Paul, 1939 Cheltenham Ave.
1957. Moyer, David G., 701 W. Main St., Lansdale, Pa.
1941. Mucklé, Craig Wright, 330 S. 9th St. (7)
1929. Mudd, Stuart, Department of Bacteriology, Univ. of Pa. School of Medicine.
1949. Mulholland, Stanford W., 22nd & Parkway.
1935. Mullen, Carroll Richard, 2025 Locust St.
1934. Mullen, Edward A., 472 Roxborough Ave.
1930. Murphy, Douglas P., 601 S. Bowman Ave., Merion Station, Pa.
1953. Murphy, Edward J., Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1958. Murphy, John J., 3600 Spruce St. (4)
1955. Murtagh, Frederick, 3701 N. Broad St. (40)
1951. Myers, David, 3701 N. Broad St. (40)
1955. Nachod, Grace R., 5501 Greene St. (44)
1945. Naide, Meyer, 2034 Spruce St.
1951. Naidoff, David, 3500 Tudor St. (36)
1950. Nardone, Anthony A., 912 S. 49th St.
1958. Nealon, Thomas F., Jr., 1025 Walnut St. (7)
1948. Neese, Paul H., 17 Derwen Road, Cynwyd, Pa.
1936. Nelson, Guy M., 255 S. 17th St.
1943. Nelson, Waldo E., 615 Moreno Rd., Narberth, Pa.
1955. Nemir, Paul, Jr., 255 S. 17th St. (3)
1905. Newcomet, William S., 3501 Baring St.
1938. Newton, Zachariah B., 601 E. Sedgwick St.
1957. Nichols, Russell Lloyd, Thomas O. Dec Memorial Hosp., 24th & Harrison Blvd., Ogden, Utah
1935. Nicholson, Jesse Thompson, 1740 Bainbridge St. (46)
1947. Noone, Ernest L., 4501 Cedar Lane, Drexel Hill, Pa.
1905. Norris, Charles C., Darby and Goshen Roads, Bryn Mawr, Pa.
1954. Norris, Charles M., 3401 N. Broad St. (40)
1905. Norris, George W., 250 S. 18th St. (3)
1938. Norris, Robert F., 513 Wynnewood Ave., Wynnewood, Pa.
1957. Oakey, Richard S., Jr., 309 Medical Bldg., Lankenau Hospital (30)
1947. O'Keefe, John J., 255 South 17th St.
1954. Olsen, Axel K., 230 N. Broad St. (2)
1913. O'Neal, Alexander Hay, St. Davids, Pa.
1949. O'Neill, James F., 140 Roslyn Ave., Glenside, Pa.

1954. O'Neill, Thomas J. E., Centennial Bldg., Front St. & Lehigh Ave. (25)
1947. Ornsteen, A. M., 2007 Delancey Pl.
1947. Ornston, Darius Gray, 36 Carpenter Lane.
1936. Orr, Theodore E., 3815 Chestnut St. (4)
1958. O'Sullivan, Ward D., Misericordia Hospital (43)
1955. Ottenberg, Donald J., 3519 N. Broad St. (40)
1913. Outerbridge, George W., 1927 Spruce St.
1949. Padis, Nicholas, 255 S. 17th St.
1954. Palmer, Louis H., Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1909. Parish, Benjamin Dores, 704 Bethlehem Pike, Flourtown, Pa.
1945. Park, Felix Roman, 1980 Utica Sq., Tulsa, Okla.
1940. Parker, Alan P., Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1948. Parkhurst, Leonard Woods, 330 S. 9th St.
1944. Paschkis, Karl E., Jefferson Medical College.
1952. Paxson, Newlin F., 250 S. 18th St.
1932. Payne, Franklin L., Univ. of Pa. Hospital.
1954. Pearce, Alexander E., 1420 Race St. (2)
1958. Pearce, Rowan C., Jr., 514 Cooper St., Camden, N. J.
1943. Pearson, Gerald H. J., Presidential Apts.—Adams House.
1954. Pearson, Manuel M., 111 N. 49th St. (39)
1952. Peck, Roy I., 3400 Spruce St.
1930. Pendergrass, Eugene P., Univ. of Pa. Hospital.
1953. Penneys, Raymond, University of Pennsylvania Hospital. (4)
1914. Pepper, O. H. Perry, 945 Parks Run Lane, Villanova, Pa.
1916. Percival, Milton Fraser, 2332 S. Broad St.
1942. Perkins, William Harvey, 1025 Walnut St.
1946. Perloff, William Harry, 3521 N. Broad St.
1951. Perri, Angelo M., 814 Federal St.
1953. Persky, Abram H., 1737 Chestnut St. (3)
1940. Pessel, Johannes F., 224 West State St., Trenton, N. J.
1952. Peters, Michael, 262 W. Broad St., Telford, Pa.
1940. Pettit, Horace, Times Medical Bldg., Ardmore, Pa.
1949. Pettit, Mary DeWitt, 3343 W. Penn St. (29)
1915. Pfeiffer, Damon B., Meadowbrook, Pa.
1946. Pfeiffer, Mildred C. J., 358 Valley Rd., Merion, Pa.
1935. Phillips, Arthur W., 3904 Walnut St.
1911. Piersol, George Morris, 308 Kent Rd., Wynnewood, Pa.
1948. Pierson, Daniel B., Jr., 740 Beacon Lane, Merion, Pa.
1957. Pilling, George Platt, IV, 2600 N. Lawrence St. (33)
1933. Pillsbury, Donald M., Univ. of Pa. Hospital. (4)
1950. Pincus, Irwin J., 269 S. 19th St.
1957. Platt, Ruth M., 441 Lyceum Ave. (28)
1951. Poinsard, Paul J., 2031 Locust St.
1937. Polk, David Stewart, Rosemont, Pa.
1956. Pontarelli, Domenic J., 255 S. 17th St. (3)
1953. Porreca, George A., 1912 Spruce St. (3)
1932. Porter, Roland De Lance, Abington Medical Bldg., Abington, Pa.
1951. Post, Joseph W., 1930 Chestnut St.
1951. Pote, Harry H., 4032 Walnut St.
1958. Potter, H. Phelps, Jr., 3400 Spruce St. (4)
1951. Pressman, Robert S., 170 W. Olney Ave. (20)
1947. Prickett, John A., Warrington, Bucks County, Pa.
1954. Prince, Leon N., 2025 Spruce St. (3)

1931. Pryor, Charles Allen, 255 S. 17th St.
1952. Pugh, James E., 614 Church Lane, Yeadon, Pa.
1947. Putney, Floyd Johnson, 1719 Rittenhouse Sq.
1958. Rachlin, Lillian, Veterans' Adm. Center, Whipple, Arizona
1943. Radbill, Samuel X., 7043 Elmwood Ave.
1953. Radbill, Sidney G., 37 S. 20th St. (3)
1945. Rakoff, Abraham Edward, 269 S. 19th St.
1953. Ralston, Edgar L., 3400 Spruce St. (4)
1931. Ramsey, Frank M., 620 E. Willow Grove Ave.
1953. Ranieri, Tito A., Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1941. Rankin, Charles A., Ludlow & Heather Rd., Upper Darby, Pa.
1946. Rapoport, Milton, Children's Hospital, 1740 Bainbridge St.
1951. Rathmell, Thomas K., 446 Bellevue Ave., Trenton, N. J.
1926. Ravdin, I. S., Univ. of Pa. Hospital. (4)
1956. Ravdin, Robert Glenn, 3400 Spruce St. (4)
1959. Raventos, Antolin, 3400 Spruce St. (4)
1954. Reagan, Lindley B., 175 Madison Ave., Mt. Holly, N. J.
1928. Reese, Warren S., 2118 Locust St.
1920. Rehfuss, Martin E., 131 St. George's Rd., Ardmore, Pa.
1921. Reiff, E. Paul, 1927 Spruce St.
1919. Reimann, Stanley P., 703 W. Phil-Ellena St.
1958. Reinhardt, David J., III, 1600 N. Broom St., Wilmington, Del.
1956. Reishtein, William A., 255 S. 17th St. (3)
1951. Rentschler, Laurence B., 5800 Ridge Ave.
1945. Rhoads, Jonathan E., Univ. of Pa. Hospital. (4)
1951. Richardson, Fred Mac D., 160 Carpenter Lane.
1919. Richardson, Russell, Phila. Tuberculosis Sanatorium, #4, Corinthian & Girard Aves.
1951. Riegel, Donald, 5110 Chester Ave.
1942. Riggs, Helena E., 150 Hewitt Rd., Wyncote, Pa.
1936. Ritter, Joseph A., 2405 N. 54th St.
1932. Robbins, Frederick R., 317 Milbank Rd., Bryn Mawr, Pa.
1952. Robbins, Robert, 3401 N. Broad St. (40)
1954. Roberts, Brooke, 3400 Spruce St. (4)
1939. Roberts, Ella, 133 S. 36th St.
1957. Roberts, Joan Mary, 4 W. Mt. Pleasant Ave. (19)
1932. Robertson, Harold F., 2118 Pine St.
1954. Robertson, Hugh, 255 S. 17th St. (3)
1938. Roche, Philip Q., Route 1, Conshohocken, Pa.
1954. Roesler, Elizabeth Mertens-, 224 W. School House La. (44)
1955. Rogers, Fred B., 3401 N. Broad St. (40)
1951. Ronis, Bernard J., 2106 Spruce St.
1948. Roscoe, Constantine R., 7226 Castor Ave.
1928. Rose, Edward, 426 Owen Rd., Wynnewood, Pa.
1936. Rose, Elizabeth Kirk, 426 Owen Rd., Wynnewood, Pa.
1959. Rose, Isadore, 6000 W. Oxford St. (31)
1952. Rose, S. Brandt, Chestnut Hill Hospital.
1946. Rosemond, George Parrott, 3401 N. Broad St. (40)
1948. Rothman, Maurice M., 1727 Spruce St.
1951. Rouse, George P., Jr., 2031 Locust St.
1952. Roxby, Bruce S., 5501 Greene St.
1955. Roxby, John B., Jr., 4821 Germantown Ave. (44)
1949. Royster, Henry P., 3400 Spruce St.
1941. Royster, Hubert A., Jr., 888 Glenbrook Ave., Bryn Mawr, Pa.

1952. Rubin, I. Edward, 255 S. 17th St. (3)
1933. Rudolphy, Jay Besson, 230 S. 21st St.
1946. Rupp, Charles, 133 S. 36th St.
1947. Rush, Alexander, 330 S. 9th St.
1945. Rynes, Samuel E., 334 S. 21st St.
1952. Sain, Fletcher D., 8 Orchard Ave., Bristol, Pa.
1958. Sall, Manuel, 255 S. 17th St. (3)
1952. Salner, Nathan P., 6812 Castor Ave. (24)
1952. Saltzman, Maurice, 1923 Spruce St.
1944. Sampson, David Alan, 726 Braeburn Lane, Penn Valley, Narberth, Pa.
1936. Sands, Joseph Evans, Rosemont, Pa.
1950. Sarner, Joseph B., 269 S. 19th St. (3)
1949. Saul, Leon J., Highland Ave., Media, Pa.
1908. Sautter, Albert C., Germantown Professional Bldg.
1954. Savacool, J. Woodrow, 146 W. Tulpehocken St. (44)
1947. Saÿen, John J., 3600 Spruce St. (4)
1948. Scarano, Joseph Albert, 1432 S. Broad St.
1917. Schaeffer, J. Parsons, 4634 Spruce St. (39)
1929. Scheffey, Lewis C., 255 S. 17th St.
1942. Scheie, Harold G., 313 S. 17th St.
1929. Schenck, Harry P., 1235 Wyngate Rd., Wynnewood, Pa.
1945. Schlezinger, Nathan S., 255 S. 17th St.
1917. Schnabel, Truman G., 1704 Pine St.
1958. Schnabel, Truman G., Jr., Phila. General Hospital (4)
1957. Schnall, Nathan, 2037 Pine St. (3)
1955. Schreader, Charles J., 318 Godfrey Ave. (20)
1958. Schreck, Kenneth M., Temple Univ. School of Medicine (40)
1952. Schulz, Norbert J., 1901 Walnut St.
1950. Schumann, Francis N., 8811 Germantown Ave.
1948. Schwarz, Gabriel A., 133 S. 36th St. (4)
1955. Schwegman, Cletus W., Hospital of the University of Penna. (4)
1930. Scott, John Porter, Children's Hospital, 1740 Bainbridge St.
1950. Scott, Michael, 255 S. 17th St.
1940. Scott, T. F. McNair, Children's Hospital, 1740 Bainbridge St.
1952. Seifer, Arthur F., 3401 N. Broad St.
1954. Seifter, Joseph, Wyeth Institute for Medical Research (1)
1957. Seipel, Russell C., 6000 Jefferson St. (31)
1951. Seitchik, Joseph N., 1417 Race St., Hahnemann Medical College Annex.
1958. Sellers, Alfred M., Hosp. of the Univ. of Pa. (4)
1952. Semisch, Charles W., III, 5329 Rising Sun Ave.
1958. Shafer, Albert H., 1004 E. Cherry Hill Apts., Merchantville 10, N. J.
1951. Shaffer, Bertram, 1825 Pine St.
1950. Sharp, Reuben L., 719 Cooper St., Camden, N. J.
1928. Sharpless, Frederick C., Beach Haven, N. J.
1958. Sharpless, Isaac, 1235 Wendover Rd., Rosemont, Pa.
1956. Shaw, Daniel L., Jr., 702 Knox Rd., Villanova, Pa.
1948. Shay, Harry, 3400 N. Broad St.
1955. Shechter, Fred R., 1923 Spruce St. (3)
1954. Shelley, Walter B., 36th & Spruce Sts. (4)
1955. Shenkin, Henry A. 255 S. 17th St. (3)
1946. Shepherd Samuel Garfield 2039 Pine St.
1932. Sheridan, Joyce T., 412 W. Durham St.
1956. Sherson, Jacob S., 131 Channing Ave., Malvern, Pa.

1936. Shipman, James Shelby, 255 S. 17th St.
1958. Shipps, Hammell P., 719 Cooper St., Camden 2, N. J.
1957. Shope, Edward Pierce Lentz, 807 Wilson Bldg., Camden, N. J.
1946. Shoup, George Daniel, 121 W. Walnut Lane (44)
1955. Shubin, Harry, 6601 N. 21st St. (38)
1952. Shuman, Charles R., 3401 N. Broad St.
1936. Shuster, Benjamin H., 1824 Pine St.
1949. Sidlick, David M., 255 S. 17th St.
1941. Silcox, Louis E., Lankenau Medical Bldg. (31)
1956. Sillars, Charles H., Abington Memorial Hospital, Abington, Pa.
1955. Silverstein, Alexander, 2114 Pine St. (3)
1954. Singmaster, Lawrence, Lankenau Medical Bldg. (31)
1952. Skromak, Stanley J., 5108 Torresdale Ave.
1958. Sloviter, Henry A., 3400 Spruce St. (4)
1929. Smith, Austin T., 1830 Spruce St.
1955. Smith, J. Winslow, 3034 Darby Rd., Ardmore, Pa.
1933. Smith, Lauren Howe, 111 N. 49th St.
1952. Smith, Richard T., 330 S. 9th St.
1927. Smyth, Calvin Mason, Abington Memorial Hospital, Abington, Pa.
1951. Snape, William J., 573 Stevens St., Camden, N. J.
1938. Snodgrass, L. E., 709 Walnut St. (6)
1953. Snyder, G. Gordon, Bryn Mawr Medical Bldg., Bryn Mawr, Pa.
1958. Sodeman, William A., 1025 Walnut St. (7)
1935. Sokoloff, Martin J., 255 S. 17th St.
1940. Soloff, Louis A., 255 S. 17th St.
1950. Somerville, William J., 2022 Locust St.
1935. Sommer, George N. J., 120 W. State St., Trenton, N. J.
1944. Sommer, George N. J., Jr., 120 W. State St., Trenton, N. J.
1952. Sones, Maurice, Mt. Airy Medical Bldg.
1951. Sonneborn, Duane G., 1200 W. Erie Ave.
1928. Spaeth, Edmund B., 1930 Chestnut St.
1958. Spaeth, Philip G., 1930 Chestnut St. (3)
1937. Spangler, John Luther, Devon, Pa.
1957. Spelman, Joseph Worcester, Dept. of Public Health Dev. Examiner, 13th & Wood Sts. (7)
1935. Spiegel, Ernest, 6807 Lawnton Ave.
1955. Spitz, Eugene B., Children's Hospital (46)
1958. Splendido, Joseph A., 608 W. Cliveden (19)
1944. Stadie, William C., University of Pennsylvania Hospital (4)
1957. Stanley, Robert C. G., 2315 Edgemont Rd., Chester, Pa.
1927. Starr, Isaac, 505 Cresheim Valley Rd.
1952. Stauffer, Herbert M., 3401 N. Broad St. (40)
1952. Stayman, Joseph W., Jr., Germantown Hospital.
1945. Stecher, H. Armin, 1510 Darby Rd., Havertown, Pa.
1955. Steel, Howard H., 3401 N. Broad St. (40)
1952. Steiger, William A., 3401 N. Broad St. (40)
1946. Stein, Irvin, 1936 Spruce St.
1953. Stein, Raymond O., 269 S. 19th St. (3)
1936. Steinfield, Edward, N. E. Cor. 18th and Chestnut Sts.
1942. Stengel, Alfred, Jr., 160 Hodge Rd., Princeton, N. J.
1951. Sterling, Julian A., 1008 Sharpless Rd., (26)
1948. Stevens, Lloyd W., 133 S. 36th St.
1925. Stokes, John H., 4228 Spruce St.
1928. Stokes, Joseph, Jr., Children's Hospital, 1740 Bainbridge St.

1943. Stokes, S. Emlen, 129 Chester Ave., Moorestown, N. J.
1954. Stone, Hrant H., E. Shawmont Ave. (28)
1952. Stoner, Emery K., 222 E. Mt. Pleasant Ave.
1953. Strawbridge, Rendall R., Lankenau Medical Bldg. (31)
1923. Stroud, William Daniel, 1011 Clinton St.
1937. Strumia, Max M., Penn Valley, Narberth, Pa.
1937. Sturgis, Margaret C., 349 Wistar Rd., Wynnewood, Pa.
1927. Sturgis, Samuel Booth, 349 Wistar Rd., Wynnewood, Pa.
1934. Summey, Thomas J., Burlington County Hosp., Mt. Holly, N. J.
1930. Sunderman, F. William, 1833 Delancey Place (3)
1936. Surver, James Miller, 1015 Walnut St.
1954. Sussman, Marcel S., 101 S. 20th St. (3)
1952. Sweeney, Francis X., 2351 E. Allegheny Ave. (34)
1955. Taggart, Harold A., 235 N. 15th St. (2)
1952. Talbot, Timothy R., Jr., 204 Booth Lane, Haverford, Pa.
1943. Tassman, Isaac S., 136 S. 16th St.
1946. Tauber, Robert, 2019 Walnut St.
1952. Taylor, Ann Gray, 6364 Germantown Ave.
1931. Taylor, Norman H., 100 W. Evergreen Ave.
1956. Templeton, John Y., III, 1025 Walnut St. (7)
1951. Theodos, Peter A., 1930 Chestnut St.
1946. Thomas, Carmen C., 1930 Chestnut St.
1952. Thompson, Charles M., 255 S. 17th St.
1927. Thorington, J. Monroe, 37 S. 20th St. (3)
1936. Thorp, Francis Quicksall, 4900 Penn St.
1936. Thudium, William J., 136 S. 16th St.
1954. Tindall, Dorothy D., 37 S. 20th St. (3)
1935. Tocantins, Leandro Maués, 1025 Walnut St.
1945. Toland, Joseph J., Jr., 1241 W. Lehigh Ave.
1935. Toland, Owen J., Episcopal Hospital, Front and Lehigh Ave.
1952. Tondreau, Roderick L., 6470 Drexel Rd. (31)
1949. Tornay, Anthony S., 37 S. 20th St.
1945. Tourish, William J., 250 S. 17th St.
1936. Towson, Charles Emory, 224 W. Washington Lane (44)
1955. Trimpi, Howard D., 1447 Hamilton St., Allentown, Pa.
1954. Trommer, Philip R., 258 S. 18th St. (3)
1956. Troncelliti, Mario V., Pennsylvania Hospital, (7)
1953. Tropea, Frank, Jr., 500 Mulberry Lane, Haverford, Pa.
1947. Trueman, Robert H., 2101 Chestnut St. (3)
1954. Truitt, R. Marshall, Jr., 740 West View St. (19)
1959. Tuddenham, William J., 3400 Spruce St. (4)
1938. Tuft, Louis, 1530 Locust St.
1933. Tumen, Henry J., 1900 Rittenhouse Sq.
1938. Turman, Christopher M., Wyncote, Pa.
1923. Turner, Creighton, H., 1731 Pine St.
1954. Turner, Linton W., 3401 N. Broad St. (40)
1929. Tyson, Ralph M., 255 S. 17th St.
1937. Uhle, Charles A. W., Lankenau Medical Bldg. (31)
1953. Ulin, Alexander, W., 230 N. Broad St. (2)
1907. Ullom, Josephus T., Carpenter and Quincy Sts.
1950. Valdes-Dapena, Antonio, 40 Lokesley Rd., Merion, Pa.
1958. Van den Noort, Gordon S., 255 S. 17th St. (3)
1937. Vander Veer, Joseph B., 330 S. 9th St.

1937. Van Loon, Emily Lois, 1930 Chestnut St.
1933. Vastine, Jacob H., 2nd, 136 S. 16th St.
1958. Velkoff, Cyril L., 2100 Walnut St. (3)
1955. Viek, Peter, 1941 Woodland Rd., Abington, Pa.
1952. Visher, Thomas J., 5903 Greene St.
1940. Voegelin, Adrian W., Germantown Professional Bldg.
1941. Vogel, Stoughton R., 151 W. Tulpehocken St.
1927. Wagers, Arthur J., 1429 Spruce St.
1949. Wagner, Frederick B., Jr., 800 Chauncy Road, Narberth, Pa.
1949. Wagner, Joseph A., 201 Highland Ave., Bryn Mawr, Pa.
1954. Wainer, Amos S., 1621 Spruce St. (2)
1938. Waldman, Joseph, 404 Meadowbrook Lane
1955. Waldron, Jerome M., 2300 Cheltenham Ave. (50)
1932. Walkling, Adolph A., 136 S. 16th St.
1955. Warner, Halsey F., 100 W. Coulter St. (44)
1956. Watkins, E. Lloyd, 8815 Germantown Ave. (18)
1954. Waugh, Elizabeth S., 348 Green Lane (28)
1952. Weaver, Harry S., Jr., 37 S. 20th St.
1955. Weaver, Robert L., 530 Walnut St. (5)
1941. Weaver, Ruth H., 2601 Parkway.
1953. Weber, Lennard L., 255 S. 17th St. (3)
1957. Webster, Marie Blanchefield, 5428 N. 5th St.
1930. Weeder, S. Dana, 250 W. Tulpehocken St.
1954. Weiner, Louis H., 4025 Girard Ave. (4)
1952. Weinstein, George L., 255 S. 17th St.
1954. Weinstock, Horace L., 329 S. 18th St. (3)
1923. Weiss, Edward, 269 S. 19th St.
1950. Weiss, Sidney, 1930 Chestnut St.
1959. Wellenbach, Burton L., 255 S. 17th St. (3)
1951. Welty, Jack W., Montgomery Ave. & Owen Rd., Wynnewood, Pa.
1955. Wendkos, Martin H., 4015 Baltimore Ave. (4)
1959. Whayne, Tom F., 36th & Spruce Sts. (4)
1935. Whelan, G. L., 2100 Walnut St.
1921. Whitaker, William, 4645 Oakland St.
1936. White, Ellen Pawling Corson-, Gladwyne, Pa.
1925. Widman, Bernard P., 250 S. 18th St.
1954. Wieder, Henry S., Jr., 330 S. 9th St. (7)
1939. Willauer, George, 6129 Greene St.
1907. Williams, Carl, School Lane and Greene St.
1937. Williams, John C., 6370 Germantown Ave.
1920. Williams, Philip F., 353 Bala Ave., Bala-Cynwyd, Pa.
1950. Williams, R. G., Univ. of Pa. School of Medicine (4)
1927. Williamson, Ernest G., 6353 Woodbine Ave.
1949. Willson, J. Robert, 3401 N. Broad St. (40)
1921. Wilson, George, 133 S. 36th St.
1945. Wilson, John F., 2013 Delancey Pl.
1953. Wilson, Julius L., Henry Phipps Institute, 7th & Lombard Sts. (47)
1937. Wilson, Ross B., Lankenau Medical Bldg. (31)
1949. Wilson, William W., 133 S. 36th St.
1957. Winkelman, N. William, Jr., 1930 Chestnut St. (3)
1948. Winston, Julius, 5839 Chester Ave.
1941. Wirts, C. Wilmer, 2017 Delancey St.
1953. Wohl, George T., 1227 Remington Rd., Wynnewood, Pa.

1933. Wohl, Michael G., 1727 Pine St.
1958. Woldow, Irving, Einstein Med. Center, York & Tabor Rds. (41)
1944. Wolf, Lewis R., 3474 Frankford Ave.
1958. Wolf, John H., 124 W. Walnut Lane (44)
1918. Wolferth, Charles C., 133 S. 36th St.
1957. Wolgamot, John R., 244 W. Main St., Moorestown, N. J.
1957. Wolkowicz, Michael I., 2022 Spruce St. (3)
1942. Wolman, Irving J., 7607 Woodlawn Ave. (26)
1893. Wood, Alfred C., 2035 Walnut St.
1950. Wood, Dorothy D., 2035 Walnut St.
1932. Wood, Francis C., 212 Laurel Lane, Haverford, Pa.
1944. Wood, H. Curtis, Jr., Skippack Pk., Whitemarsh, Pa.
1953. Woodruff, Marston T., 4940 Penn. St. (24)
1928. Wright, Carroll S., 1402 Spruce St.
1921. Ylvisaker, Lauritz S., Fidelity Mutual Life Ins. Co., Parkway at Fairmount, (30)
1941. Young, Barton R., Germantown Hospital
1953. Zarafonetis, Chris J. D., 3401 N. Broad St. (40)
1957. Zaslow, Jerry, 1351 Tabor Rd. (41)
1955. Zatuchni, Jacob, 3401 N. Broad St. (40)
1946. Zeigerman, Joseph H., 2105 Spruce St. (3)
1949. Zintl, William J., 69th & Market Sts., Upper Darby, Pa.
1956. Ziserman, Abraham J., 2046 Pine St. (3)
1933. Zulick J. Donald, Abington Memorial Hospital, Abington, Pa.

Non-Resident Fellows

ELECTED

1912. Addison, William H. F., 286 East Sidney Avenue, Mt. Vernon, N. Y.
1947. Allbritten, Frank F., Jr., Univ. of Kansas Medical Center, Kansas City 3, Kansas
1941. *Aronson, Roland S., Walter Reed Army Hospital, Washington 12, D. C.
1954. Austin, George M., University of Oregon Medical School, Portland, Oregon
1934. Bank, Joseph, 800 N. First Ave., Phoenix, Arizona
1931. Bates, William, Harrisburg Hospital, Harrisburg, Pa.
1932. Behney, Charles A., Dept. of Health, Old DeWitt Rd., Lansing, Mich.
1949. Bixby, Edward W., Casey Key, R.D. Box 31-13, Nokomis, Fla.
1951. Blank, Harvey, Jackson Memorial Hospital, Miami 36, Fla.
1937. Braceland, Francis J., The Institute of Living, 200 Retreat Ave., Hartford, Conn.
1951. Brinton, S. Jervis, 115 92nd St., Stone Harbor, N. J.
1936. Brown, Charles L., 591 Montgomery St., Jersey City (2)
1948. *Brown, Robert B., U. S. Naval Hospital, Bethesda, Md.
1953. Burbidge, J. Raymond, 163 Nassau St., Princeton, N. J.
1957. Butterworth, Thomas, 411 Walnut St., Reading, Pa.
1908. Cadbury, William W., 274 W. Main St., Moorestown, N. J.
1948. Chapman, John P., Calle Fray Junipero Serra 26, Palma de Mallorca, Balearic Islands, Spain.
1950. Chapple, Charles C., 1530 - 16th St., Washington, D. C.
1941. Cheleden, John, 133 Broadway, Daytona Beach, Fla.
1930. Clarke, Francis Mann, 116 New St., New Brunswick, N. J.
1926. Clerf, Louis H., 5575–8th Ave., North, St. Petersburg, Fla.
1930. Cottrell, James E., McGuire Veteran's Administration Hospital, Richmond 19, Va.
1940. Crowe, Aldrich C., 1208 Ocean Ave., Ocean City, N. J.

* Military service.

1947. Cutler, Norman L., 1300 Harrison St., Wilmington, Del.
1941. *Cuttle, Tracy D., U. S. Pacific Fleet Post Office, San Francisco, Calif.
1955. Daugherty, J. Arthur, 610 N. Third St., Harrisburg, Pa.
1954. Deitrick, John E., Cornell Medical College, 1300 York Ave., New York 21, N. Y.
1941. Dixon, Harold M., 4805–46th St., N.W., Washington, 16 D. C.
1959. Durgin, Bernice Elise, 221 N. Allegheny St., Bellefonte, Pa.
1955. Edwards, Margaret Hay, 224 W. State St., Trenton, N. J.
1951. Ellis, Mackinnon, Veteran's Hospital, White River Junction, Vt.
1957. *Errion, Arthur Robbins, U. S. Naval Hospital, Key West, Fla.
1951. Ervin, Carl E., 240 North Third Street, Harrisburg, Pa.
1944. Estes, W. L., Jr., 35 E. Elizabeth Ave., Bethlehem, Pa.
1954. Evans, Harry D., Jr., American Cyanamid Co., 1 Caspar St., Danbury, Conn.
1952. Faller, Constantine P., 127 State St., Harrisburg, Pa.
1940. Forrester, James S., Harrisburg Polyclinic Hospital, Harrisburg, Pa.
1951. Friedland, Carl K., 271 W. Blackwell St., Dover, N. J.
1947. Gaskill, Herbert S., 250 Ash St. Denver, Colorado.
1913. Gates, Nathaniel, 10 Peterboro, Detroit, Mich.
1957. Giering, John Fleming, 84 James St., Kingston, Pa.
1931. Gilman, Robert Louis, Cheyney, Pa.
1954. Godfrey, Ellwood W., Princeton Hospital, Princeton, N. J.
1928. Gordon, Burgess L., The Lovelace Foundation, Albuquerque, New Mexico.
1952. Grieco, Reynold M., 2 W. 4th St., Williamsport, Pa.
1952. Gross, Elmer R., Medical Arts Bldg., Wilmington, Del.
1933. Gruber, Charles M., 1313 College Ave., Redlands, Calif.
1955. Harris, John H., 1301-A N. Second St., Harrisburg, Pa.
1936. Hatch, Lerleen Clement, 2184 Ayres Ave., Akron, Ohio.
1928. Hayman, Joseph Marchant, Jr., Tufts College Medical School, Boston, Mass.
1944. Hershey, J. Ivan, VA Hospital, Shreveport, La.
1955. Hills, Arthur G., 1000 N. W. 17th St., Miami, Fla. (36)
1933. Hitzrot, Lewis H., Medical Director, Mercersburg Academy, Mercersburg, Pa.
1950. Horn, Robert C., Henry Ford Hospital, Detroit, Mich.
1938. Howell, John C., 1104 Logan Ave., Tyrone, Pa.
1936. Hughes, Brady A., 312 Euclid Ave., Mt. Brook, Birmingham, Ala.
1953. Imber, Irving, 428 Walnut St., Reading, Pa.
1958. Impink, Robert R., 405 Oley St., Reading, Pa.
1935. Ingleby, Helen, The Church House, Heacham, King's Lynn, England
1921. Ivy, Robert H., 104 Dalton Rd., Paoli, Pa.
1952. Jones, Ralph, Jr., Jackson Memorial Hospital, Miami, Fla.
1955. Jordan, Charlotte B., 519 Sarah St., Stroudsburg, Pa.
1955. Jordan, Claus G., 519 Sarah St., Stroudsburg, Pa.
1956. Keffer, William H., 413 Oley St., Reading, Pa.
1947. Kelchner, Clyde H., 1125 Turner St., Allentown, Pa.
1944. LaMotte, William O., Brook Hill Farms, Hockessin, Del.
1954. LaMotte, William O., Jr., Professional Bldg., Wilmington 3, Del.
1952. Levan, John B., 401 Oley St., Reading, Pa.
1949. Lhamon, William T., Baylor University College of Medicine, Houston, Tex.
1954. Light, John J. B., 25 N. 9th St., Lebanon, Pa.
1954. Lindenmuth, Woodrow W., VA Hospital, West Haven, Conn.
1948. Livingood, Clarence S., Henry Ford Hospital, Detroit 2, Mich.
1933. Long, Esmond R., Conifer, Pedlar Mills, Va.
1919. Lynch, Frank B., Jr., U. S. Veterans Hospital, Coatesville, Pa.
1948. McCall, Milton L., Louisiana State Univ., New Orleans, La.
1954. McCrory, Wallace W., Dept. of Pediatrics, University Hospital, Iowa City, Iowa

1937. McGuinness, Aims C., Room 5238, Dept. of Health, Education & Welfare, Washington, D. C.
1948. McLaughlin, John T., 1252 S. Central Ave., Glendale, Calif.
1949. Manlove, Francis R., 6015 E. 17th Ave., Denver, Colo.
1953. Margolies, Michael, 567 E. Chestnut St., Coatesville, Pa.
1955. Margolies, M. Price, 567 E. Chestnut St., Coatesville, Pa.
1935. Mason, James B., 40 E. Erie St., Chicago 11, Ill.
1932. Meade, Richard H., Jr., 750 San Jose Dr., Grand Rapids, Mich.
1936. Middleton, William Shainline, Veterans' Administration, Washington, D. C.
1946. Miller, Franklin R., Snyder-Jones Clinic, First Nat. Bk. Bldg., Winfield, Kansas.
1928. Moon, Virgil H., Univ. of Miami, Coral Gables, Fla.
1949. Motley, Hurley L., 1212 Shatto St., Los Angeles, Calif.
1950. Neefe John R., 627 Eleventh St., St. Petersburg, Fla.
1937. Nicholson, Samuel T., 2d., 642 High St., Pottstown, Pa.
1941. Nisbet, Verner, 714 Queens Rd., Charlotte 7, N. C.
1956. Oppenheimer, Albert, 224 E. Main St., Morestown, N. J.
1926. Paul, John Rodman, Dept. Preventive Medicine, Yale University, New Haven, Conn.
1947. Pearce, Louise, Trevenna Farms, Orchard Rd., Skillman, N. J.
1959. Pendergrass, Henry P., Massachusetts General Hospital, Boston 14, Mass.
1922. Pleasants, Henry, West Chester, Pa.
1953. Raffensperger, Edward C., 2039 N. 2nd St., Harrisburg, Pa.
1938. Read, Hilton S., 5407 Atlantic Ave., Atlantic City, N. J.
1936. Reisinger, John A., 901 20th St., N. W., Washington, D. C.
1955. Rettew, Philip L., 612 Museum Rd., Reading, Pa.
1947. Rogers, Arthur Merriam, 1351 Durwood Drive, Charlotte, N. C.
1907. Royer, B. Franklin, 429 E. Baltimore St., Greencastle, Pa.
1956. Rubenstone, Albert I., Michael Reese Hosp., Chicago, Ill.
1954. Sampson, Martin C., Keydross Lane, Saratoga, N. Y.
1953. Scanlan, Robert L., St. Vincent's Hospital, 2131 W. 3rd St., Los Angeles 57, Calif.
1945. Schell, James F., 139 W. Maple Ave., Langhorne, Pa.
1928. Schofield, Frederick S., Veteran's Administration Hospital, Fort Wayne, Ind.
1921. Schumann, Edward A., "Henneberg", Chalfont, Pa.
1913. de Schweinitz, George L., 3231 Arroyo Chico, Tucson, Ariz.
1941. Shands, Alfred R., Jr., Alfred I. duPont Institute, Rockland Rd., Wilmington, Del.
1908. Shannon, Charles E. G., 9 Park St., Waterville, Maine.
1950. Shiffer, Paul H., Dreher Ave., R.D. #2, Stroudsburg, Pa.
1951. Shorey, Winston K., Jackson Memorial Hospital, Miami, Fla.
1932. Siegel, Alvin Erdreich, 464 W. Buford Rd., Macon, Ga.
1933. Smelzer, Donald C., Lancaster General Hospital, Lancaster, Pa.
1954. Smith, Charles W., 121 State St., Harrisburg, Pa.
1929. Sponsler, Marshall B., 103 Lansdowne Road, DeWitt, N. Y.
1953. Steele, Brandt F., 717 Pontiac St., Denver 20, Colo.
1952. Steele, Eleanor, 717 Pontiac St., Denver 20, Colo.
1955. Stevenson, Theodore D., Presbyterian Board of Foreign Missions, 156 Fifth Ave., New York 10, N. Y.
1934. Stewart, Sloan G., Pacific and North Carolina Avenues, Atlantic City, N. J.
1931. Stiles, Merritt H., 104 5th Ave., Spokane 4, Wash.
1949. Stockwell, Eunice L., Tryon, N. C.
1953. Stroud, Morris W., 3rd, Harvard Rd., Cleveland, Ohio
1944. Swenson, Paul C., The Charles T. Miller Hospital, 125 W. College Ave., Saint Paul 2, Minnesota
1928. Thomas, J. Earle, College of Medical Evangelists, School of Medicine, Loma Linda, Calif.
1942. Thompson, Wesley D., 247 E. 3rd St., Lewistown, Pa.
1941. Tompkins, Pendleton, 450 Sutter St., San Francisco, Calif.
1897. Veasey, Clarence A., 1118 W. 9th Ave., Spokane 9, Wash.

1933. Waygood, James L., Pocono Pines, Pa.
1936. Wilder, Theodore S., Beckley Memorial Hospital, Beckley, W. Va.
1935. Willard, John H., Harlan Memorial Hospital, Harlan, Kentucky.
1957. Witkow, Alexander, 401 Columbia Road, Hamilton, Ohio
1952. Wood, Horatio C., III, 5319 Prospect Rd., Peoria, Ill.
1947. Zintel, Harold A., St. Luke's Hospital, Amsterdam Ave. & 113th St., New York 25, N. Y.

Honorary Fellows

Limited to Fifty, of Whom Twenty May Be Foreigners

American

ELECTED

1917. Miner, Charles Howard, B.S., M.D., Member of Board of Directors, Wilkes-Barre General Hospital; Board of Health, Wilkes-Barre; Board of Directors, White Haven Sanatorium Association and Penna. Tuberculosis Society. 264 S. Franklin St., Wilkes-Barre, Pa.
1936. Richards, Alfred Newton, Ph.D., Sc.D., M.D. (hon.), LL.D., Emeritus Professor of Pharmacology, School of Medicine, and Associate Trustee, University of Pennsylvania.
1942. Russell, Frederick Fuller, M.D., Brig. Gen. Med. Corps, U.S.A. (Ret.). Lime Kiln Lane, Route 1, Box 488R, Louisville, Kentucky.
1958. Shryock, Richard H., Ph.D., American Philosophical Society, Independence Square (6)
1947. Stone, Robert Spencer, M.D., University of California Medical School, San Francisco, Cal.

Foreign

1937. Dale, Sir Henry Hallett, C.B.E., F.R.S., M.A., M.D., F.R.C.P., 52 Queen Anne St., London, W. 1, England.
1922. McCarrison, Sir Robert, Kt., C.B.E., M.D., D.Sc., LL.D., F.R.C.P., Major-General, Indian Medical Service (retired). 18 Linton Rd., Oxford, England.
1955. Mann, Ida, M.A., D.Sc., M.B., B.S., L.R.C.P., D.O.M.S., F.R.C.S. Ophthalmic Consultant to the Government of W. Australia, 56 Hobbs St., Nedlands, Perth, W. Australia.

Honorary Associate Fellows

1955. Clement, Martin W., 1964 Surburban Station Bldg. (3)
1955. Diemand, John A., President, Insurance Company of North America, 1600 Arch St. (2)

Necrological List

Resident Fellow	*Date of Election*	*Date of Death*
Aronson, Joseph D.	March 4, 1942	October 17, 1958
Colgan, Robert C.	January 8, 1934	June 7, 1958
Corrigan, Patrick H.	October 3, 1945	May 28, 1958
Creighton, William J.	June 2, 1920	September 22, 1958
Creskoff, Adolph S.	January 6, 1941	January 21, 1959
Donnelly, Edward J.	January 7, 1942	October 11, 1958
Godfrey, Lincoln	January 7, 1953	December 17, 1958
Jackson, Chevalier	October 10, 1917	August 16, 1958
Jonas, Leon	June 5, 1918	September 29, 1958
Kelly, Thomas C.	November 6, 1912	April 21, 1958

Resident Fellow	*Date of Election*	*Date of Death*
Lieberman, Louis M.	October 13, 1948	January 19, 1959
Loewenberg, Samuel A.	May 11, 1931	December 4 1958
McConnell, James W.	May 6, 1935	May 8, 1958
Moorhead, Stirling W.	January 2, 1924	April 26, 1958
Morris, Elliston J.	January 4, 1893	August 31, 1958
Roberts, Walter	December 6, 1899	March 31, 1958
Rodman, John Stewart	January 3, 1912	April 26, 1958
Strecker, Edward A.	October 3, 1923	January 2, 1959
Tait, Edwin Forbes	October 13, 1948	September 23, 1958
Tucker, Gabriel	January 11, 1926	April 17, 1958
Weidman, Fred D.	June 2, 1920	August 30, 1956
Wood, Horatio C.	April, 1865	March 31, 1958
Zentmayer, William	March 7, 1894	March 18, 1958
Non-Resident Fellows		
Guthrie, Donald	January 6, 1936	October 30, 1958
McCarthy, Daniel J.	December 5, 1900	October 9, 1958
Vastine, Mary Frances	April 2, 1947	December 11, 1958
Vaux, Norris Wistar	January 3, 1912	August 19, 1958

(Suggested Form of Bequest)

I hereby give, devise, and bequeath to The College of Physicians of Philadelphia, a charitable and educational association organized under the laws of the Commonwealth of Pennsylvania and the executive offices whereof are located in the City of Philadelphia, the sum of.....

a. which sum shall be used for the general purposes of said association;

b. which sum shall be added to and become a part of the general endowment fund of said association;

c. which sum shall be used by said association preferably for the following purposes:

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